Annual Meeting • San Francisco • June 21-25, 1954

This Issue Exceeds 165,000 Copies

THE JOURNAL

OF THE

American Medical Association

MARCH 20, 1954

ORIGINAL ARTICLES

PAUL EHRLICH: HIS CONTRIBUTIONS TO MEDICINE 969
H. C. S. Aron, M.D., Chicago
TREATMENT OF URINARY TRACT INFECTION 972
D. Parker, M.D., Seattle
CONTROL OF URINARY INCONTINENCE IN PATIENTS WITH MULTIPLE SCLEROSIS 975
S. R. Muellner, M.D., Boston
NOCTURNAL AND INSULIN GASTRIC SECRETION 977
W. H. Olson, M.D., and A. B. Bridgwater, M.S., Chicago
VERTEBRAL FRACTURES AS A COMPLICATION OF ELECTROCONVULSIVE THERAPY 981
P. A. Dewald, M.D.; N. M. Margolis, M.D., and H. Weiner, M.D., Rochester, N. Y.

CLINICAL NOTES

CARDIAC ARREST AFTER INTRAVENOUS ADMINISTRATION OF PROCAINE AMIDE (PRONESTYL) 985
W. Weingarten, M.D.; N. J. Galluzzi, M.D., and A. A. Doerner, M.D., Staten Island, N. Y.
SURGICAL REPAIR OF INTRAVENTRICULAR SEPTAL DEFECTS 986
E. B. Kay, M.D., and H. A. Zimmerman, M.D., Cleveland
CONTRACTION OF LEFT LEAF OF DIAPHRAGM COINCIDENT WITH CARDIAC SYSTOLE 987
A. Sjoerdsma, M.D., Ph.D., Bethesda, Md., and W. B. Gaynor, M.D., Brooklyn
MACROSCOPIC DIAGNOSIS IN LIVER BIOPSY 990
R. B. Terry, M.D., London
DEFORMITY OF THE XIPHOID CARTILAGE ASSOCIATED WITH GASTRO-INTESTINAL SYMPTOMS 992
C. R. Hanlon, M.D., Bethlehem, Pa., and M. M. Miller, M.D., New York
EPITHELIOMA AND FIBROSARCOMA OF THE PENIS OCCURRING SIMULTANEOUSLY 993
J. H. McDonald, M.D., and N. J. Heckel, M.D., Chicago
DIFFICULTIES IN EARLY DIAGNOSIS OF MYOSITIS OSSIFICANS 994
L. Weinstein, M.D.; S. H. Fraerman, M.D., and P. Lewin, M.D., Chicago
INVERTED APPENDICAL STUMP SIMULATING CANCER OF THE CECUM 996
A. M. Vaughn, M.D., and J. Widran, M.D., Chicago
FATAL HEMORRHAGE FOLLOWING NEEDLE BIOPSY IN UREMIA 997
S. Zelman, M.D., Topeka, Kan.
USE OF A TILT-TABLE IN EARLY REHABILITATION OF PARAPLEGIC PATIENTS 1000
S. Climo, M.D., New Haven, Conn.

SPECIAL REPORTS

Council on Pharmacy and Chemistry

NEW AND NONOFFICIAL REMEDIES 1001

Council on Physical Medicine and Rehabilitation

APPARATUS ACCEPTED 1003
AN APPRECIATION 1003

Council on Foods and Nutrition

ACCEPTED FOODS 1003

EDITORIALS

Building the Program for the Scientific Assembly 1004
Employment for Diabetics 1005
Illegal Operation of Medical Diathermy Apparatus 1005

ORGANIZATION SECTION

Texas Medical Association 1006
Council on Industrial Health 1006
Students' Annual Convention May 1-3 in Chicago 1006
More "March of Medicine" TV Shows 1007
Audio-Digest Profits Go to Medical Schools 1007
Federal Medical Legislation 1007
State Medical Legislation 1007

REGULAR DEPARTMENTS

Medical News 1008
Meetings 1013
Examinations and Licensure 1015
Deaths 1016
Foreign Letters:
Australia: Australia's National Health Service 1020
Ecuador: Hemodynamic Changes in Anemia. Blood Groups in Indians of Ecuador. Antihistaminic Agents for Burns 1021
Finland: Campaign Against Tuberculosis. Crude Liver Extracts for Cirrhosis of the Liver 1021
Israel: Side-Effects of Penicillin. Scholarships for Medical Students 1022
Turkey: Idiopathic Anemia 1022
Correspondence:
Deceleration and the Motor Car. Chlorophyll. Hemophilia and Clotting Time. Sanatorium Treatment for Tuberculosis. International Health. Tonsils and Adenoids 1023
Miscellany:
Federal Communications Commission Rules Regulating the Operation of Medical Diathermy Equipment. Milliequivalents. 1026
Business Practice:
The Physician and His Reception Room 1027
Medicolegal Abstracts 1027
Medical Literature Abstracts 1028
Book Reviews 1044
Queries and Minor Notes:
Discoid Lupus Erythematosus. Hirsutism at the Climacteric. Numbness of Hands in Pregnancy. Poliomyelitis at a Summer Camp. Chronic Ulcerative Colitis. Small Tumor Near Hyoid Bone. Hand-Schüller-Christian Disease. Glucose Solution Intramuscularly 1047
State Medical Associations ..... Adv. Page 6

WASHINGTON NEWS

State Health Grants. "Doctor-Draft" Act. Hill-Burton Funds. Medical Research. Pesticides. ..... Adv. Page 13
Tonics and Sedatives ..... Adv. Page 54
The Bright Side ..... Adv. Page 67
Books Received ..... Adv. Page 74
Index to Advertisers ..... Adv. Page 78

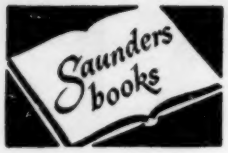
Volume 154, No. 12 Published Weekly at 535 North Dearborn Street, Chicago 10, Illinois. Subscription, $15. Single Copy, 45 cents

New (2nd) Edition—**Mayo Clinic Diet Manual**

This conveniently arranged manual describes every standard diet in use at the Mayo Clinic today. The general composition of each diet, its adequacy, foods included and excluded, and sample menus are discussed.

See SAUNDERS Advertisement on next 2 pages

ARTHUR C CURTIS MD
UNIVERSITY HOSP
ANN ARBOR MICH
1529290 MAR 20 011



if you ever see a urinary tract infection . . . .

Davis' Mechanisms of Urologic Disease

If you ever see a urinary tract infection, Doctor, or a prostatitis, you'll appreciate the common-sense help this book gives you.

Dr. Davis has distilled his vast experience to present a plan of *effective approach to urologic disease*. Discarding many time-worn theories and concepts, he presents a simple formula for investigating and treating this group of disorders —a group that *can* be very troublesome.

The author explains the obstructive-infectious concept, points out that practically all urologic disorders affect the *entire* urinary tract, calls *chronic prostatitis* a "mere pathologic term, exalted fallaciously into a disease." As he proceeds, the entire subject comes into sharp focus and you see clearly how the underlying disturbed physiology of these disorders may be returned to normal.

You'll like this book.

By David M. Davis, M.D., Professor of Urology Emeritus, Jefferson Medical College; Visiting Lecturer in Urology, Graduate School of Medicine, University of Pennsylvania. 156 pages, 5½" x 8½", illustrated. $4.50. *New!*

a book of wonderfully practical hints . . . .

Dunphy & Botsford's Examination of the Surgical Patient

Emphasis in this popular book is on plain, old-fashioned clinical observation, with scores of experience-proven hints on *how to make greatest use of your unaided senses.*

The presentation is strictly in the "how-to" vein. The authors tell you what to look for, how to look for it, and what your findings mean in terms of pathology. They tell you exactly *how* to feel the thyroid gland; *how* to recognize early cancer of the mouth; *how* to appraise an acute head injury; *how* to differentiate an acute abdomen; *how* to distinguish anatomical anomalies from pathology. And they alert you, with equal precision, to the *pitfalls* in physical examination.

The brilliantly practical section on *The Emergency Examination* is alone worth the price of the book.

By J. Englebert Dunphy, M.D., F.A.C.S., Associate Clinical Professor of Surgery, Harvard Medical School; Surgeon, Peter Bent Brigham Hospital; Consultant in Surgery, Children's Medical Center; and Thomas W. Botsford, M.D., F.A.C.S., Clinical Associate in Surgery, Harvard Medical School; Senior Associate in Surgery, Peter Bent Brigham Hospital; Associate in Surgery, Children's Medical Center. 326 pages, 6" x 9", 188 illustrations. $7.50.

a "road map" through pelvic surgery . . . .

Parsons & Ulfelder's Atlas of Pelvic Operations

Only *the currently useful technics* in pelvic surgery are included in this new atlas. Some 95 procedures are presented, each one in such brilliant detail that the book is actually a "road map" through pelvic operations.

With large, beautifully drawn illustrations and the concise instructions that accompany them, the authors take you through each step in each operation. As they proceed, they point out the exact spot in each procedure where "trouble" is most likely to occur, and explain what to do about it if it is encountered.

Surgery for malignancy is covered with great thoroughness. The authors emphasize that such technics differ markedly from the procedures employed for benign lesions in the identical organs.

By Langdon Parsons, M.D., Professor of Gynecology, Boston University School of Medicine; Chief, Department of Gynecology, Massachusetts Memorial Hospital; Gynecologist, Palmer Memorial Hospital; Gynecologist, Massachusetts Department of Public Health, Hospital for Cancer at Pondville, Massachusetts; and Howard Ulfelder, M.D., Assistant Clinical Professor of Gynaecology, Harvard Medical School; Assistant Surgeon, Massachusetts General Hospital; Gynecologist, Massachusetts Department of Public Health, Hospital for Cancer. Illustrated by Mildred E. Codding. 321 pages, 14" x 11½", with 207 plates. $18.00. *New!*

Use convenient **SAUNDERS** Order Form on next page ➡

THE JOURNAL of the American Medical Association is published weekly by the American Medical Association. Subscription price, $15.00 a year. 45c a copy. Canadian $16.50. Foreign $19.00. Entered as second class matter June 25, 1885, at the Postoffice at Chicago, Ill., under act of March 3, 1879. Address all communications to American Medical Association, 535 N. Dearborn St., Chicago 10, Illinois

THE JOURNAL
of the American Medical Association
Published Under the Auspices of the Board of Trustees

VOL. 154, NO. 12 — CHICAGO, ILLINOIS — MARCH 20, 1954
COPYRIGHT, 1954, BY AMERICAN MEDICAL ASSOCIATION

PAUL EHRLICH: HIS CONTRIBUTIONS TO MEDICINE

A TRIBUTE AT THE CENTENNIAL OF HIS BIRTHDAY

Hans C. S. Aron, M.D., Chicago

Most great men in medicine are remembered because their names are connected with one fundamental discovery, with the description of one or several diseases, with important symptoms or signs, or with technical or operative procedures. Paul Ehrlich's contributions to medicine are so numerous that it is not possible to link his name with one particular achievement. In hematology, in immunology, and in chemotherapy (a means of treatment which he originated), Ehrlich established many basic facts, developed a number of fundamental methods, and coined a multitude of terms that have become the common property of the medical profession. They are used so frequently that the name of Paul Ehrlich now is seldom quoted in this connection. At the centennial of the birthday of this genius and pioneer, it appears to be appropriate to recollect those of his accomplishments that have withstood the test of time to the present day.

Paul Ehrlich was born March 14, 1854, in Strehlen, a small town in Silesia, Germany. A brilliant scholar at the gymnasium and an exceptional student in medicine, he had already started laboratory work of his own before graduation. At the age of 23, Ehrlich published his first scientific paper; he described a new type of cell that he had found, which he called *Mastzellen* or mast cells, a term used to this day. The discovery of the mast cells was the first practical result of Ehrlich's attempts to utilize the aniline dyes, recently created by the chemical industry, for staining purposes in histological studies. While he was a resident in internal medicine at the University Hospital in Berlin, called the Charité, he developed the basic methods used in modern hematology.

He devised the technique of preparing a thin layer of blood and applying the most suitable dyes to this smear when dry. All available dyes of various chemical character and many different mixtures of them were tested. For the first time Ehrlich studied the granulations in the protoplasm of the white blood cells. He found and characterized the eosinophils, the basophils, and the neutrophils (later called the polymorphonuclear white blood cells). He described, furthermore, the myelocytes, the large mononuclear leukocytes, and the transitional leukocytes. He showed how to distinguish three types of red blood cells—the normoblasts, the megaloblasts, and the microblasts or poikiloblasts. With his command of Greek and Latin, Ehrlich easily found a characteristic name for each type of blood cell that he discovered. Most of these terms were universally accepted and are used unchanged to the present day. In 1887, Ehrlich showed how it was possible from a drop of blood to recognize lymphatic leukemia and to distinguish it from the bone marrow cell leukemia, later called myeloid leukemia. In the following year, Ehrlich described a new type of anemia, which he called aplastic anemia, and claimed as the cause of this anemia, "no activity of the bone marrow."

Ehrlich's profound knowledge of the chemical procedures used in the synthesis of organic dyes led him to find a special test in urine, the so-called diazo reaction. Of much farther reaching importance was Ehrlich's wide experience with organic dye products for developing staining methods to recognize and to identify the recently discovered bacteria. He introduced methylene blue for this purpose and recommended this dye to his friend, A. Neisser, for use in Neisser's successful search for the organism that causes gonorrhea.

On March 24, 1882, Robert Koch had reported before the Physiological Society in Berlin that he had discovered the bacillus that caused tuberculosis, which he called the "tubercle bacillus." Less than six weeks later, on May 1, 1882, Ehrlich demonstrated before the Society of Internal Medicine in Berlin his method of staining and identifying Koch's tubercle bacillus by means of its acid-fastness, which Ehrlich had discovered. Ehrlich's method for staining the tubercle bacillus, described in 1882, is practically the same used everywhere today. Robert Koch gave greatest credit to Paul Ehrlich: "It is due to Ehrlich's staining method for finding the presence of the tubercle bacilli that the search for tubercle bacilli in the sputum was universally adopted. Without Ehrlich's method but just a very few research workers may have been able to study the tubercle bacillus." Thus wrote Robert Koch in 1883.

From the Department of Pediatrics, Northwestern University Medical School, and the Children's Memorial Hospital.
Dr. Hermann Pinkus, Wayne University College of Medicine, Detroit, furnished reprints, abstracts, and a memoir concerning Paul Ehrlich.

While Ehrlich intensively searched for tubercle bacilli in the various clinical forms of tuberculosis, he fell seriously ill in 1888. As tubercle bacilli were found in his own sputum, he had to interrupt all his activities. Accompanied and guarded by his wife, Ehrlich went to Egypt for nearly two years for rest and recreation. In 1890, Ehrlich returned fully recovered to Berlin. During this period of relaxation Ehrlich's genius had conceived new ideas. His interest was now attracted by problems of immunity. His investigations were started with methods not applied before. Ehrlich did not use substances of bacterial origin but used instead well-characterized toxic protein products of higher plants, such as ricin and abrin. He showed how animals could be immunized by injection of increasing doses of these toxic substances. He devised methods to determine quantitatively the degree of immunity and the amount of antitoxins present in the blood or serum of the immunized animals. He showed that immunized nursing mice are able to transfer their specific immunity to their offspring but that no immunity can be transferred from immunized father animals. While fascinating from a biological point of view, these studies appeared to the medical profession to be without any practical interest. Few suspected that the results of Ehrlich's "theoretical" studies shortly would revolutionize their therapeutic activities and help them to save many of their patients.

In 1892, Emil von Behring had succeeded in immunizing horses against diphtheria with diphtheria toxin and in producing a completely new type of remedy, a horse serum containing diphtheria antitoxin. However, Behring recognized that the degree of antitoxic efficacy of his products could not be controlled. Before it would be possible to use this new product for the treatment of diphtheria in human beings, it was imperative to find some way to measure the curative potency of the various serums obtained from immunized horses. At this critical moment, Behring sought the advice and the cooperation of Paul Ehrlich.

On the basis of his experience, Ehrlich with his collaborators, Kolle and von Wassermann, developed procedures of obtaining potent antitoxic serums. Ehrlich devised exactly defined units, which he called I. U. (immunization units), for the determination of the content of antitoxins, and he established the fundamental methods of standardizing antitoxin in these immunization units, which were accepted in all civilized countries. Finally, the dose of antitoxin required for the treatment and for prophylaxis of diphtheria was determined in Ehrlich's immunization units. The introduction of diphtheria antitoxin, a scientifically standardized remedy against a dreadful disease, had an enormous impact on medical and especially on pediatric practice. The demand for this unique product rose enormously in Europe, America, and other continents.

In 1896, the Prussian government erected in Steglitz, then a suburb of Berlin, a new institution called "Royal Institute for Standardization and Investigation of Antitoxic Sera." The 42-year-old extraordinary professor of internal medicine at the University of Berlin, Paul Ehrlich, was made the director of this institute and was given the title *Geheimer Medizinalrat* (Privy Medical Counsel). Here, Ehrlich completed his outstanding monograph on the "Standardization of Diphtheria Antitoxin." He also stipulated the two fundamental principles of immunology: 1. Toxins are strictly specific. 2. Toxins introduced into the animal organism cause the production of equally specific antitoxins, far in excess of the amount required to neutralize the amount of toxin introduced.

To illustrate the reaction between toxins and the body cells and between toxins and antitoxins, Ehrlich developed his much-discussed "side chain theory." According to this theory, each toxin molecule has two groups, the toxophore group that carries the toxic action and the haptophore group that fits into a receptor group of the cell. When the receptor groups of a cell are filled with haptophore groups, the cell is stimulated to produce new receptor groups; according to the law of hyper-regeneration, these new receptor groups are produced in excessive numbers and therefore are released from the cell into the circulation, where they act as antibodies. During these studies Ehrlich made the observation that, after certain procedures, toxins in solutions could lose their toxophore groups but retain their haptophore groups. Ehrlich called these derivatives of the original toxins "toxoids." He emphasized that these toxoids had lost their toxicity but were still able to stimulate the production of antitoxins. He showed how it is possible to transform solutions of toxins into solutions of the corresponding toxoids, and he recommended these toxoids for the purpose of immunizations. It was a quarter of a century and later before the fundamental value of Ehrlich's "toxoids" finally was recognized and the use of toxoids for immunization against diphtheria and tetanus became a universally accepted procedure.

While an increasing number of specific antitoxins were used with great enthusiasm all over the world, Paul Ehrlich recognized that immunotherapy had its definite limitations. It was not possible at that time to prepare antitoxic products against the protozoa, such as the Plasmodium malariae, the trypanosomes, spirochetes, and spirillas, nor against the newly discovered filtrable viruses. Ehrlich felt that, in the search to combat these parasites, he had to return to his old trail, to substances that through their chemical structure had a specific affinity to the parasites and were, in Ehrlich's nomenclature, "parasitotrop." His new slogan was "We must learn to aim chemically!"

Toward the end of the 19th century, larger facilities were planned for Ehrlich's work, to be erected in Frankfurt am Main. According to Ehrlich's suggestions, this new institution was built as the "Royal Institute for Experimental Therapy." The examination and standardization of the antitoxins was confined to one department; the greater part of the laboratories was devoted to the search for chemical products suitable for curing infectious diseases. To indicate that this was an entirely new way, different from the methods pharmacology had used up to this time, Ehrlich coined the term "chemotherapy." The best definition of the principles of chemotherapy is given in Ehrlich's own words: "To be successful in chemotherapy we must search for substances which are fitted to annihilate the invading parasites with the least possible damage to the host organism."

For his chemotherapeutic studies Ehrlich then resorted to experimental infections with trypanosomes and spirochetes. These parasites were transmitted to large series of mice, rats, rabbits, and other laboratory animals to test the curative action of any number of chemotherapeutic agents. As such, Ehrlich chose at first his beloved aniline dyes but soon turned more and more to organic compounds of arsenic, which were systematically synthesized according to his designs. The series of animals and the number of preparations tested rose to gigantic figures. His staff of co-workers grew steadily, especially after 1906 when a second institution, the "Georg Speyer House," was built adjacent to the institute. The 606th arsenical compound, arsphenamine (Salvarsan), proved to be a remedy that gave therapeutic results far exceeding those of any drug ever used before in the treatment of syphilis and several tropical diseases. The discovery of arsphenamine in 1910 was regarded as an unprecedented triumph of chemotherapeutic research. Ehrlich was overloaded with honors and decorations.

At the 17th International Congress of Medicine held in London in 1913, Ehrlich gave a summarizing address on chemotherapy. With great satisfaction he told how in Surinam, Dutch Guiana, a hospital for the treatment of 300 patients suffering from frambesia (yaws) had to be closed because this disease, caused by Spirochaeta pertenuis, a relative of Spirochaeta pallida, could now be successfully cured after one injection of arsphenamine. When arsphenamine had been replaced by the easier-to-handle neoarsphenamine (Neosalvarsan), the treatment of syphilis with this drug was generally introduced in several European countries and systematically controlled. Here the incidence of syphilis declined progressively, and, by the middle of the 1930's, congenital syphilis in infants had become almost unknown until the population shifts during World War II brought occasional reappearances.

As important as these therapeutic results appeared at their time, the inspiration that Paul Ehrlich gave to the further development of research and therapy was much farther reaching and longer lasting. His fascinating personality and the unique facilities in both institutions under his direction attracted numerous scientists from many countries. From the United States may be mentioned these men: Professor Herter from New York; Reid Hunt from Harvard, who was Ehrlich's assistant for two years; and William H. Welch, the great reformer of medical education in this country, from Johns Hopkins. A wider part of the medical profession was stimulated by the numerous lectures that Ehrlich was invited to give: the Croonian and the Harben Lectures in England, and the Herter Lectures in New York and Baltimore, followed by lectures in Washington and Chicago. He addressed congresses in Paris, Brussels, and London. In December, 1908, in Stockholm, after being awarded the Nobel Prize, Ehrlich gave an acceptance speech entitled "Partial Functions of the Cell."

Many of the ideas that Ehrlich developed and followed experimentally from 1904 to 1914 concerned the same problems that today, after more than 40 years, form the center of research and interest in chemotherapy. Early Ehrlich recognized and showed experimentally the dangers of too sudden and too forceful destruction of the parasites because of the toxic products that may be liberated. He emphasized that it is fully sufficient for his "therapia sterilisans magna" to make "each individual parasite unable to reproduce." An ideal chemotherapeutic agent, according to Ehrlich, prevents the multiplication of the parasites. Today we have learned that the action of our best sulfonamides and antibiotics is bacteriostatic and not bactericidal.

In several instances Ehrlich recommended his "combination therapy," the simultaneous application of several chemotherapeutic agents with an entirely different chemical structure and therefore with different "chemoceptors" to attack the parasites from various sides. Ehrlich showed experimentally that in such a combination the toxic side-effects to the host are simultaneously reduced. Again at the International Congress in London in 1913, Ehrlich reported the great success that was achieved by using a combination of arsphenamine with acriflavine (Trypaflavine) and *o*-chlororosaniline (Tryparosan) for the treatment of trypanosomiasis in the Congo region of Africa. Today the same principle is applied in the chemotherapeutic treatment of tuberculosis. Here small doses of the somewhat toxic streptomycin are combined with *p*-aminobenzoic acid (PABA) and isoniazid products.

Ehrlich's greatest concern was the problem of drug-fastness. He recognized as the most frequent cause of drug-fastness treatment with doses that are nearly but not fully sterilizing. He emphasized again and again the dangers of under-treatment. He reported on a strain of arsenic-fast trypanosomes, which, in his laboratory during three years, was transmitted from mice to mice in 400 passages, and the last strain was still arsenic-fast. Ehrlich found that strains from cases in which relapses occurred were not infrequently fast to the drug that had been used for treatment previously. Drug-fastness is mostly limited to one type of drug, but, by prolonged and alternated under-treatment with three different trypanocidal agents, Ehrlich was able to produce a strain of trypanosomes fast to all three chemotherapeutic drugs. It appears that Ehrlich, 40 years ago, because of his extensive studies, was more aware of the dangers of drug-fastness than we realize today.

At Ehrlich's 60th birthday, March 14, 1914, 37 scientists published, as *Festschrift*, a monumental volume of 668 pages entitled "Paul Ehrlich, Eine Darstellung seines wissenschaftlichen Wirkens" (a description of his scientific achievements). Less than five months later World War I broke out. All research was completely disrupted. The man whose endeavor through all his life had been to alleviate human suffering felt his ideals to be wrecked and ruined. Fate spared him still greater disappointments. Paul Ehrlich died on Aug. 20, 1915. His mortal remains were interred in the Jewish Cemetery of Frankfurt, but the results of his genius have survived. During the first 20 years after his death, chemotherapeutic research produced new and improved remedies for malaria, sleeping sickness, and other tropical diseases, in addition to those of minor importance. The world-moving success came when Gerhard Domagk, following orthodoxically the trail shown by the great

pioneer, combined Ehrlich's favored azo dyes with the amide of sulfanilic acid, which had been the model for Ehrlich's arsanilic acid. Domagk's product Prontosil (2,4-diaminoazobenzene-4′-sulfonamide) proved to be the first chemotherapeutic agent of high potency against many of the bacteria that cause the most frequent infectious diseases in children. A disagreeable side-effect of Prontosil, the discoloration of the skin, was removed after Fourneau and his group at the Institute Pasteur in Paris had shown that the azo dye component was not absolutely necessary for the therapeutic effect. Prontosil album (sulfanilamide) was introduced and became the mother substance of the numerous sulfonamide drugs synthesized in the following years, the amount now numbering nearly 5,000.

What role Paul Ehrlich's ideas played in the development of penicillin, the first antibiotic, was recently revealed by the very best informed person. In 1902, a young British physiologist, Dr. Henry Hallet Dale, went to Frankfurt and worked with Paul Ehrlich for two years. Ehrlich took a great liking to this gifted scholar who later became one of Britain's most prominent scientists, was awarded the Nobel prize, and became Sir Henry Dale. As director of the British National Institute for Medical Research, he was informed of each and every step that led to the development of penicillin. On the basis of his experience, Sir Henry Dale in 1949 wrote as follows: "In 1935 Domagk's discovery of the action of 'Prontosil' opened the way to the series of the Sulphanilamide derivatives; and the revolutionary improvements which these effected in the treatment of a number of common infections, played its part in the decision of Florey and his team to examine anew the chemotherapeutic possibilities of the half-forgotten Penicillin, which Fleming had found ten years before. And the new enterprise by the marvelous success of Penicillin has now led to the discovery of Streptomycin, and others are already in sight. But the central idea of experimental chemotherapy, and the initial impulse to its triumphant march forward, came from Paul Ehrlich."

3837 Broadway (13).

TREATMENT OF URINARY TRACT INFECTION

Dean Parker, M.D., Seattle

Urinary tract infections are second in frequency only to infectious processes of the upper respiratory tract.[1] Because of their frequency they are often dismissed as minor problems and may be treated in a rather haphazard manner, but this attitude may lead to serious difficulty and aid in establishing chronic infections with permanent and irreparable renal damage. Contributing to this attitude has been the advent of the chemotherapeutic and antibiotic drugs, which, after a preliminary course of therapy, may give relief of symptoms with striking and immediate results. In any given case, general care and good urologic management will be of greater benefit than any chemotherapeutic treatment. Womack and his coworkers have stated that age and systemic factors not only predispose patients to urinary infection but also militate against optimum beneficial results.[2] To take the greatest advantage of the recent advances in the use of chemotherapeutic agents in urology, one should consider these important factors[3]: (1) the suitability of the patients for treatment, (2) the bacteria involved in the infectious process, and (3) the agent or agents best suited for both the patient and the bacteria present.

SELECTION OF PATIENTS FOR UROLOGIC STUDY

When the patient is seen routinely by his physician in the office, the examination may reveal signs or symptoms of urinary tract infection by means of either the history or the physical findings. Certainly all of these patients do not require complete urologic study; neither should they all be placed immediately on a chemotherapeutic regimen without further study. Those patients in whom physical findings suggest no pathological condition of the urinary tract, in whom no abnormalities are seen on a flat film of the kidney, ureter, and bladder, and in whom a simple, primary, acute infection exists caused by only one organism are the patients who are entitled to immediate treatment with one of the chemotherapeutic agents, without further urologic study. Those patients, however, who have had recurrent or chronic infections, in whom smear or culture tests reveal mixed organisms, in whom there are abnormalities of the lower urinary tract, prostate, genitals, renal mass, or in whom calcified areas that might be in the upper urogenital tract appear on the flat film are the ones who should have a complete urologic examination prior to institution of therapy. It is well known that, in the presence of abnormality of either the upper or lower urinary tract, chemotherapy is ineffective and may lead only to making the organisms resistant to the agent that, at a later date, might prove most effective. Any such urinary tract abnormality must first be corrected before more than a temporary effect can result from medication. In those cases in which it is felt that further urologic examination is necessary, the diagnostic procedure should be followed in an orderly manner, step by step, either to a satisfactory conclusion in diagnosis or to a complete urologic study with all its components. In those cases of simple acute infection in which antibiotic therapy has been instituted, if there is no immediate response, further urologic study is indicated.

Read before the Section on Urology at the 102nd Annual Meeting of the American Medical Association, New York, June 4, 1953.

1. Jawetz, E.: Practical Bacteriologic Aspects of Urinary Tract Infection, Am. Pract. & Digest Treat. **3**: 361-364 (May) 1952.

2. Womack, C. R., and others: Terramycin Therapy of Urinary Tract Infections, A. M. A. Arch. Int. Med. **89**: 240-257 (Feb.) 1952.

3. Hinman, F., Jr.: Use of Antibiotics in Urology, Ann. West. Med. & Surg. **4**: 829-833 (Dec.) 1950.

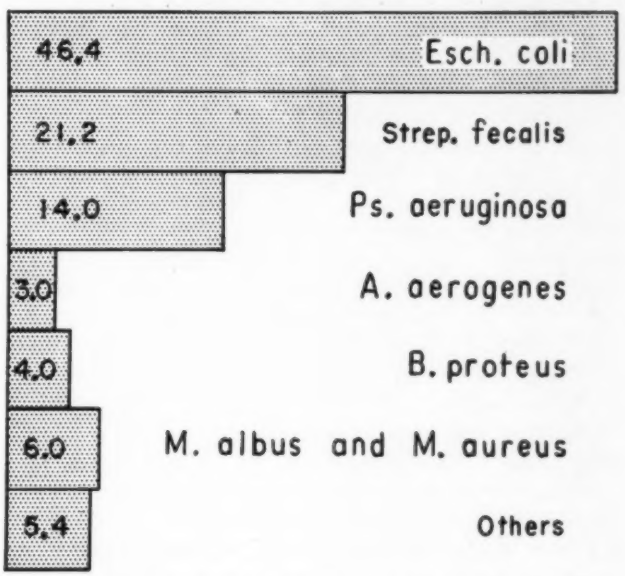
CAUSATIVE ORGANISM

After ruling out or correcting fundamental abnormalities of the urinary tract, one must consider the type and degree of infection. It is important to obtain a sterile voided specimen from a man and a catheterized specimen from a woman during an acute attack. The centrifuged specimen should be examined for bacterial and cellular content. The question of whether to prepare a smear or a culture is sometimes difficult to decide. The smear has certain advantages in that it gives an immediate result and it establishes to some degree the number of organisms involved. The culture, on the other hand, although time consuming and somewhat more expensive, is advantageous in that it insures absolute identification of the organism, and the plated culture permits the use of sensitivity studies for the organism found. The culture of the organism is necessary for the purposes of correlation and for the treatment of complicated cases. Relatively few kinds of organisms are observed in cases of urinary tract infection. Simple infections of the acute variety are usually due to Escherichia coli or Aerobacter aerogenes and a Staphylococcus (Micrococcus); after instrumentation or surgery, Proteus and Pseudomonas organisms may be found, in addition, among the rods, and Streptococcus fecalis or enterococcus organisms among the cocci.[4] It seems obvious, therefore, that, for the routine clinical application, the gram stain is of considerable value in the diagnosis of the acute simple urinary tract infection. In the complicated or recurrent or chronic case, culture and sensitivity studies should be done. There are three main reasons for using the sensitivity studies [5]: (1) chronic or recurrent infection, (2) infection resistant to previous antibiotic therapy, or (3) severe and fulminating infection.

THE ANTIBACTERIAL AGENT

For the intelligent use of the antibacterial agents in the treatment of urinary tract infection, one should consider certain principles in their action upon the living organism. The first factor to be remembered is that the action of the antibacterial agent is on the organism and not on the host. It affects the organism by interfering with certain metabolic activities of the growing cell. Most antibiotic agents are bacteriostatic in low concentrations and bactericidal in high concentrations. They differ from antiseptics in that they interfere with growing cellular metabolism rather than destroy the cell wall of both the bacteria and the host. The chemotherapeutic ratio is determined by the number of metabolic processes the agent inhibits. The second factor to be considered in the use of antibacterial agents is that the resistance of the organism to antibiotic agents may limit their effectiveness. Organisms may have natural immunity to the drug or may have acquired immunity by previous exposure. It is for this reason that it is important to have new drugs at hand, since with time and use a once-effective agent may become almost useless. This is now the case with penicillin in some instances, and the development of erythromycin has been extremely timely.

A third factor that must be considered in the use of these drugs is that toxicity may limit the dosage used. The principal limiting factor in the use of chemotherapeutic agents is toxicity, caused either by the dose relationship or by the sensitivity of the host. Many of the toxic reactions for the sulfonamide drugs were due to dose relationship, while all those from penicillin result from sensitivity of the host, with no relationship to the dosage administered. O'Conor [5] repeats an old saying, "One man's food is another man's poison," which is particularly true in this regard. Raper reports that probably 300 antibiotics have been isolated. Most are too toxic for general use, and only six or eight have reached clinical maturity. A fourth factor in considering the agent to be used is the amount of absorption by blood and tissues and excretion. Because of the relationship of the concentration to the effectiveness of the agent, this consideration is important. Consideration also should be given as to whether the process is an interstitial type of infection or a topical or mucous membrane infection. On this basis one might seek a high tissue absorption level or a high excretion level, depending on the nature of the infectious process.



Percentage distribution of 332 micro-organisms isolated from urinary tract infections in 325 patients (adapted from Rhoads et al.).

In treating interstitial infections, intensive and protracted treatment of the patient is often necessary and a thorough follow-up with urine cultures should be made despite cessation of symptoms. As an example, a child with chills, fever, and pyuria is seen by the pediatrician; treatment results in marked clinical improvement. The patient may next be seen with "honeymoon pyelitis" by the gynecologist, and brief antibiotic treatment results in an apparent cure. At 20 she is seen in the third trimester of pregnancy with signs of urinary tract disease with or without toxemia. At 30, when she again notices signs of urinary tract infection, she is seen by a urologist who finds essentially normal pyelograms but intrinsic renal infection. He starts a course of treatment with such marked improvement that she fails to return for follow-up treatment. This patient at 35 begins to feel weak and tired and

4. Rantz, L. A.: Infections of Urinary Tract, in Advances in Internal Medicine, edited by Steele, J. M., New York, Interscience Publishers, Inc., 1942, vol. 1, pp. 137-167.

5. O'Conor, V. J.: Urinary Antiseptics: Past and Present: Fact and Fancy, Proc. Inst. Med. Chicago **19**: 187-199 (Dec. 15) 1952.

again has a urinary tract flare-up. She is then found to have irreversible renal damage and uremia with chronic pyelonephritis. The prognosis is poor. This woman will require continued palliative medical care. This type of story, treatment, and result can in many cases be avoided by prolonged, intensive, and varied treatment and follow-up studies.[5a]

The last and fifth factor to be considered in the choice of an antibiotic agent is the effective drug combination. It may have a synergistic action, as in the case of streptomycin and one of the sulfonamide drugs, or an inhibiting effect such as reported with streptomycin and chloramphenicol [6] (Chloromycetin) in certain types of infection in which the combination is less effective than either drug alone.

In considering the inhibiting effect of one drug upon another, one also should consider inhibition of organisms by other organisms. This established bacterial relationship may be abolished by antibiotics. With the elimination of one group of organisms, an organism that was not pathogenic previously with normal flora may become pathogenic and cause infection.[7] An example of this is seen with the use of any of the broad spectrum antibiotics in sterilization of the intestine prior to surgery. The result may be an overwhelming staphylococcic enteritis, several cases of which have been fatal. The following combinations of drugs can be used in infectious processes due to each one of the organisms listed [8]: (1) Esch. coli, chlortetracycline (Aureomycin), oxytetracycline (Terramycin), or chloramphenicol plus sulfisoxazole (Gantrisin); (2) Str. fecalis, chlortetracycline, oxytetracycline, or chloramphenicol plus sulfisoxazole or penicillin; (3) Pseudomonas aeruginosa, oxytetracycline or streptomycin plus sulfisoxazole; (4) Proteus organisms, chloramphenicol plus sulfisoxazole; (5) A. aerogenes, oxytetracycline, chloramphenicol, or streptomycin plus sulfisoxazole; (6) micrococci, chlortetracycline plus penicillin; (7) Str. hemolyticus, penicillin plus sulfisoxazole; and (8) Klebsiella pneumoniae, chloramphenicol or streptomycin. In review of these factors, many variables are seen that might influence the effectiveness of an agent.

TOPICAL USE OF ANTIBIOTICS

While it seems rational to depend upon tissue and urinary concentrations of antibiotics in eradication of infectious disease, there does seem to be a place in the urologic armamentarium for the topical use of these agents. Certain chronic infections of the urethra or bladder causing pathological conditions that cannot be corrected, may show marked improvement with therapy with these drugs. In wound infections in which the organism is resistant to nontoxic agents, a more toxic agent may prove beneficial as a topical medication. I am thinking here of the use of 1% to 5% solutions of the broad spectrum antibiotics in nonspecific anterior urethritis and the use of polymyxin in 1% solution in kidney and bladder infections by Ps. aeruginosa or the use of bacitracin in wound infections with resistant organisms.

CONCLUSIONS

There has been a tendency to disregard well-established, somewhat standardized aspects of the treatment of urinary tract infections because of rather miraculous progress in the chemotherapeutic treatment of these diseases. Proper diagnosis, sound surgical principles, and elimination of other foci of infection plus sound supportive medical measures are still of primary importance in the treatment of these patients. Only on this basis can we expect maximum and permanent eradication of infection with careful and judicious use of any and all of the available and indicated chemotherapeutic drugs. To complete bacteriological knowledge, some trial-and-error use of the drugs may still be required before cure is attained, but new drugs are important and should be accepted for use if they are less favorable to resistance, less toxic to the patient, and more potent either in themselves or in combination with other agents than existing antibiotics or other agents.[9] Finally, the most important factors in the treatment of urinary infection are proper consideration of etiology and diagnosis, sound urologic treatment of any pathological condition discovered, good general medical management, eradication of other foci of infection,[10] and bacteriological choice of the proper drug. It is then necessary to avoid toxicity, treat fully, and follow the patient for recurrence before eradication is presumed.

1330 Madison St. (4).

5a. Birchall, R. R.: Responsibility of Interest in Treatment of Pyelonephritis, J. Urol. **68**: 798-803 (Nov.) 1952.

6. Jawetz, E.; Gunnison, J. B.; Speck, R. S., and Coleman, V. R.: Studies on Antibiotic Synergism and Antagonism: Interference of Chloramphenicol with Action of Penicillin, A. M. A. Arch. Int. Med. **87**: 349-359 (March) 1951.

7. Jawetz.[1] Sloss, P. T.: Mayo Clinic Report.

8. O'Conor.[5] Pulaski, E. J., and Baker, H. J.: Combined Therapy of Infectious Disease, South. M. J. **42**: 765-769 (Sept.) 1949.

9. Waksman, S. A., and Lechevalier, H. A.: Neomycin, Science **109**: 305-307 (March 25) 1949, cited by Duncan, G. G., and others: Neomycin: Results of Clinical Use in Ten Cases, J. A. M. A. **145**: 75-80 (Jan. 13) 1951.

10. Wilson, W. M.; Barnes, R. W., and Bergman, R. T.: Chloromycetin in Treatment of Bacillary Urinary Infections, Urol. & Cutan. Rev. **55**: 206-208 (April) 1951.

Polymixin B.—[The Drug] was used to treat ambulatory, asymptomatic and symptomatic individuals found to have *Pseudomonas aeruginosa* in the gastrointestinal tract. In the control group of asymptomatic carriers, about half of the cases were spontaneously free of the organism in the follow-up studies 25 and 30 days later. One hundred fifty milligrams of the drug were given orally every eight hours for seven days to the experimental group. After five days, all the stools were negative for pseudomonas. Stool cultures were repeated in 23 cases, 25 and 30 days after the drug had been started. At this time, all but one were negative for pseudomonas. About half of the subjects receiving the drug had mild abdominal cramps or mild diarrhea or both between the first and third days of drug administration, and these lasted from a few hours to two days. They disappeared in each instance in spite of continuation of therapy and without administration of other drugs. Polymyxin B should be considered promptly as one of the control measures in any outbreak of diarrhea in a newborn nursery where pseudomonas is found as the most likely pathogen. Its use may also be considered as a prophylactic measure when personnel are found to harbor this organism. Oral polymyxin B is very effective in eliminating *P. aeruginosa* when it is present in the gastrointestinal tract either in carriers or in active infections.—G. Y. Mills, M.D., and B. M. Kagan, M.D., Effect of Oral Polymyxin B on *Pseudomonas Aeruginosa* in the Gastrointestinal Tract, *Annals of Internal Medicine*, January, 1954.

CONTROL OF URINARY INCONTINENCE IN PATIENTS WITH MULTIPLE SCLEROSIS

S. Richard Muellner, M.D., Boston

It has been estimated that there are several hundred thousand persons in the United States who have multiple sclerosis. Recent surveys have shown that there are more than 4,500 cases of multiple sclerosis in the New England states alone, and of these 2,000 exist in Massachusetts.[1] About one-third of the patients who have multiple sclerosis have distressing urinary symptoms. These symptoms consist of frequency, nocturia, urgency, incontinence, and enuresis. In the past, very little could be done to mitigate these symptoms. Therapeutic failure was, in large measure, due to attempts to restore bladder control by drugs that were used to influence the central nervous system lesion of multiple sclerosis.

During the past three years I have had the opportunity to observe 85 patients with multiple sclerosis who in addition had urinary symptoms. The results of this study form the basis of this report and suggested that it was possible to approach the problem from another direction. To be sure, nothing could be done to alter the central nervous system lesion. Would not, however, a better understanding of the pathological physiology of micturition in multiple sclerosis enable one to influence favorably the behavior of the bladder? This shift in emphasis from the central nervous system to the bladder itself proved profitable. Better understanding of the behavior of the bladder in multiple sclerosis made it possible to allay frequency and nocturia and to enhance significantly urinary control.

Space does not permit a detailed review of the normal physiology of micturition and the way in which it is altered in multiple sclerosis.[2] For voluntary initiation of the urinary stream, however, the coordinated function of the thoracic diaphragm, the abdominal musculature, and especially the levator ani is necessary. Control of the levator ani and the pubococcygeus, which is the anterior portion of the levator ani, is needed to stop the urinary stream and to inhibit the contracting detrusor.

The patient with multiple sclerosis who suffers from urinary incontinence cannot readily contract the levator ani. He is just as awkward in the control of the levator ani as he is in the coordination of his leg muscles, an awkwardness that results in his peculiar gait. The detrusor too can be quite uncoordinated in its contraction and is extraordinarily hyperirritable. Portions of the bladder may contract separately, or the whole organ may go into spasm in response to minor stimuli (fig. 1 through 4). The commonest stimulus to induce an unwanted detrusor contraction is a sudden increase in intra-abdominal pressure that results from efforts such as rising from the recumbent or sitting posture, lifting weights, climbing stairs, and stepping onto or off curbs. These sudden detrusor contractions are responsible for the precipitous urgency and the involuntary micturition in multiple sclerosis. Because he is unable to contract the levator ani quickly, as a normal person can, the patient with multiple sclerosis cannot inhibit the detrusor that then proceeds to empty the bladder. A clear understanding of these factors that affect micturition in multiple sclerosis makes it possible to control urinary symptoms satisfactorily.

MANAGEMENT

The management of the bladder in multiple sclerosis is based on relatively simple principles. 1. The bladder must be shielded against unnecessary stimuli to reduce urgency. 2. Detrusor hyperirritability must be reduced to eliminate incontinence. 3. The bladder capacity must be increased to lessen frequency and nocturia.

Clinically, these principles can be translated into the following plan of management. The clinician must become familiar with the patient's pattern of living. This he can readily do by reviewing the patient's activity in a typical day. Since urgency and incontinence are apt to be brought on by efforts that tend to increase intra-abdominal pressure suddenly, the history should bring out which activity is most apt to be followed by sudden urgency or incontinence and ways in which such activities can be circumvented or eliminated. Information on the daily fluid intake is important. For the patient with multiple sclerosis, a large fluid intake is undesirable and needlessly increases urinary frequency. The behavior pattern of the bladder is next ascertained by a simple record, which the patient is asked to keep for a 24 hour period, in which the amount of urine in each micturition and the time of voiding are recorded. The record may show a urinary frequency of 8 to 20 times a day; nocturia of 7 to 8 times; and a voiding volume of 15 to 90 cc. The analysis of this record often shows bouts of frequency during certain hours of the day. This information is helpful in planning drug therapy.

The physical examination of the patient must include a careful rectal examination in which particular attention is paid to the tonicity of the rectal sphincter and to the ability of the patient to contract or relax the levator ani at will. The better the voluntary control of the levator ani, the easier it is, as a rule, to restore nearly normal micturition.

The neurogenic bladder of multiple sclerosis is subject to spontaneous infections. The upper urinary tract, on the contrary, is seldom affected by the disease. Acute and

---

From Harvard Medical School, Tufts College Medical School, and Beth Israel Hospital.

Read before the annual meeting of the Massachusetts Medical Society, May 21, 1953, Boston.

This study was made possible by the cooperation of the Massachusetts Chapter of the National Multiple Sclerosis Society.

The Belladonna used in this study was supplied by Sandoz Chemical Works, Inc., New York 14, and the Octin by Bilhuber-Knoll Corporation, Orange, N. J.

1. Ipsen, J., Jr.: Personal communication to the author. Ipsen, J., Jr.: Prevalence and Incidence of Multiple Sclerosis in Boston, 1939-1948, Arch. Neurol. & Psychiat. **64**: 631-640, 1950. Schumacher, G. A.: Multiple Sclerosis and Its Treatment, J. A. M. A. **143**: 1059-1065 (July 22) 1950; **143**: 1146-1154 (July 29) 1950; **143**: 1241-1250 (Aug. 5) 1950.

2. Muellner, S. R.; Loman, J., and Alexander, L.: Urinary Bladder in Multiple Sclerosis, J. Urol. **68**: 230-236, 1952. Muellner, S. R.: Physiology of Micturition, ibid. **65**: 805-810, 1951. Muellner, S. R.: Physiologic Components of Urinary Bladder, New England J. Med. **241**: 769-772, 1949.

chronic cystitis constitutes an added powerful stimulus to the hyperirritable bladder. It is, therefore, very important to determine whether the urine is infected. All therapy will fail as long as cystitis persists. Since infections are apt to recur, they must be watched for and treated vigorously with appropriate antibiotics.

The hyperirritability of the bladder can be significantly lowered or completely eliminated for hours at a time by the judicious use of anticholinergic drugs. Of these atropine sulfate is the cheapest and often the best. How much atropine to use and when to use it must be determined for each patient. Moreover, since multiple sclerosis is subject to remissions and exacerbations, the dosage schedule has to be adjusted according to the course of the disease. Successful control of urgency and incontinence does not mean that the patient must receive continuous anticholinergic medication. To do so may cause urinary retention

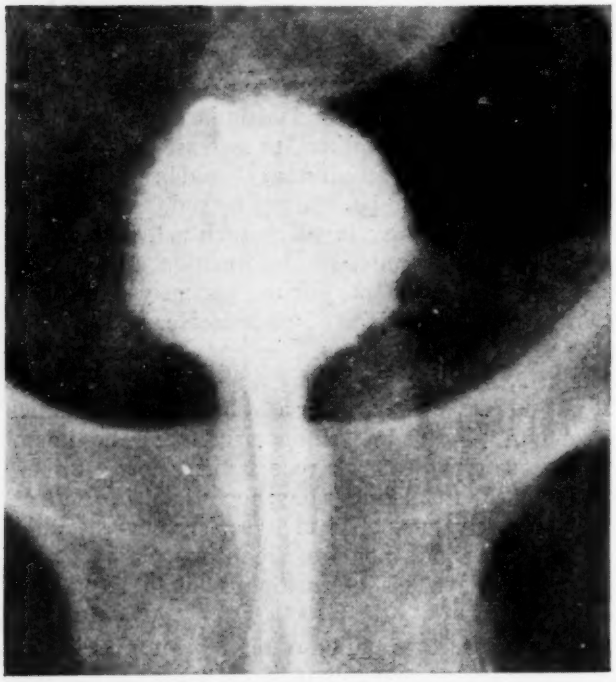


Fig. 1.—Cystogram showing hyperirritability of detrusor in patient with multiple sclerosis; note spasm of bladder from mere contact with catheter.

or relax the detrusor to the point where micturition becomes difficult or inefficient. Conversely, rule of thumb administration of the drug at arbitrarily chosen regular intervals may fail to produce urinary continence at the time when the patient most needs it. Anticholinergic drugs do not "cure" the abnormal bladder in multiple sclerosis. They can be used very successfully, however, to produce the right effect at the right time; in other words, one uses them not like an iron pill three times a day after meals but like a cocktail that is useful for a desired effect at a particular moment.

It is here that the micturition record mentioned above and the pattern-of-living information can be a very useful guide in the spacing of the medication. A tablet containing 0.4 mg. (1/150 grain) of atropine sulfate taken by mouth may abolish vesical irritability for from two to four hours. If, in the morning, the patient has a good deal of frequency and urgency that interferes with his activity, the tablet can be given one-half to three-quarters of an hour before arising. Some patients need it only for periods of the midafternoon or evening. It is easy to teach a patient to note the length of time it takes for the drug to take effect and the duration of this effect. He soon learns to take the drug in a way that will achieve urinary con-

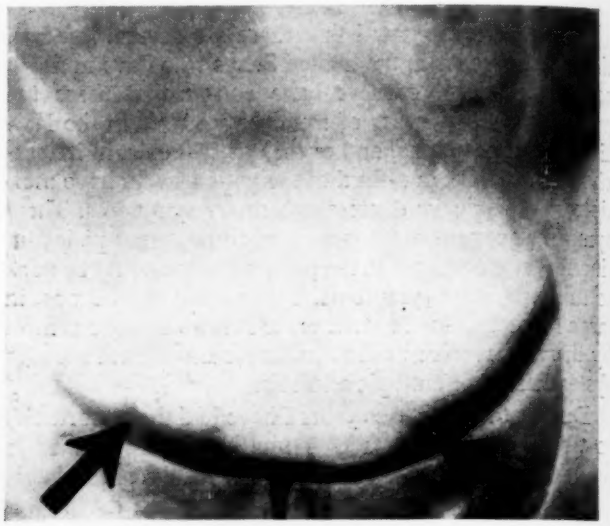


Fig. 2.—Cystogram obtained during excretory urography; Arrows point to deep folds that are results of contractions of bladder muscle.

tinence for the desired period of time. It is seldom necessary to use more than three to five tablets a day. Some patients do not tolerate atropine well because of excessive dryness of the mouth or visual disturbances. (Preexisting blurred vision may become aggravated.) When this occurs, other anticholinergic drugs may be tried. Bellafoline (containing 0.25 mg. total levorotatory belladonna alka-

Fig. 3.—Retrograde cystogram in patient with multiple sclerosis showing irregularly contracting detrusor with upward projection of bladder dome and pseudodiverticula.

loids per tablet) is a satisfactory substitute for atropine and is less apt to cause visual disturbances or dryness of the mouth. In general, it is a more comfortable drug for the patient than atropine, although it is not quite as powerful, tablet for tablet, as the latter is in allaying detrusor

hyperirritability. Another drug that can be quite useful if neither atropine nor Bellafoline is tolerated is Octin (containing 0.12 gm. [2 grains] of methylisoactenylamine per tablet). Octin also is not likely to cause visual disturbances or dryness of the mouth. Occasionally it will

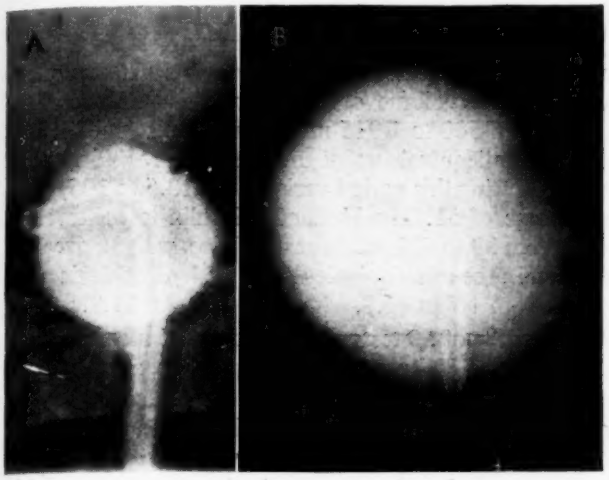


Fig. 4.—*A*, small spastically contracted bladder despite gentle filling at low pressure; *B*, same bladder with larger capacity and smooth bladder wall 30 minutes after administration of atropine.

brilliantly decrease bladder irritability. Some patients, however, experience peculiar "sensations" that they cannot describe accurately and that cause them to refuse to take the drug. Methantheline (Banthine)[3] has also been reported to have a powerful effect on the bladder and it undoubtedly has. It has been difficult, however, to achieve an even effect on bladder irritability with methantheline. It either relaxes the bladder too much or not enough. The newer derivative of methantheline, propantheline (Pro-Banthine) bromide, was not given to any of the patients.

Except in the final stages of the disease when an atonic overdistended bladder with overflow incontinence is commoner is it ever necessary to treat the urinary incontinence by means of an indwelling catheter. In some patients, the whole urinary picture may be complicated by coexisting urologic disease, such as an obstructing prostate in a man or urinary stress incontinence in a woman. The management of these problems, however, is not within the scope of this paper.

SUMMARY AND CONCLUSIONS

There are more than 4,500 cases of multiple sclerosis in the New England area; of these, 2,000 exist in Massachusetts. One-third of these patients suffer from distressing urinary symptoms, most of which can be successfully controlled by a plan of management in which the patient's pattern of living and the condition and behavior of his bladder are carefully considered. Anticholinergic drugs play an important part in the therapeutic plan, if they are used on an individual basis with due consideration of the various factors discussed.

520 Beacon St.

3. Lapides, J., and Dodson, A. I., Jr.: Observations on Effect of Banthine in Urological Disturbances, read before the Section on Urology of the American Medical Association, Chicago, June 11, 1952. Draper, J. W.; Wolf, S.; Murphy, J. F., and Kravetz, H.: Effect of Banthine in the Treatment of Genito-Urinary Disorders, J. Urol. **69**: 632-640, 1953.

NOCTURNAL AND INSULIN GASTRIC SECRETION

William H. Olson, M.D.
and
Anna B. Bridgwater, M.S., Chicago

This study on normal inmates and inmates with duodenal ulcer in a penitentiary was undertaken to settle the question of whether an ulcer patient secretes more gastric juice than a normal person. Previous studies by other investigators have demonstrated that most duodenal ulcer patients secrete a higher nocturnal volume of gastric juice with a higher acidity than normal persons. Chalfen, in 1928, reported that normal subjects do not secrete acid during the night, in contradistinction to high acid secretion in ulcer patients. Winkelstein, in 1935, confirmed these findings; 45% of his normal subjects did not reveal any free hydrochloric acid. On the other hand, Sandweiss and associates,[1] in 1946, and Brown and coworkers,[2] in 1952, found no significant difference between the nocturnal secretion of normal persons and ulcer patients, when no retention was present in the latter group. A historical review of the literature on gastric secretion in normal persons and ulcer patients was presented by Sandweiss and associates [3] and a critical evaluation of nocturnal secretion studies by Ivy and others.[4]

In our series, 97 secretion studies were made on 66 subjects—53 on 26 ulcer patients and 44 on 40 normal subjects. Studies in which subjects withdrew before the tests were completed are not included. A few tests were invalidated by technical difficulties, such as obstruction of the Levin tube or its propulsion into the duodenum as determined by fluoroscopy.

PROCEDURE

The study was performed over a period of two years. All patients were men whose average age was 40 years, the variation being from 28 to 57 years. The majority of

From the Department of Gastro-Intestinal Research, Medical Research Institute of Michael Reese Hospital.

Dr. H. Necheles gave assistance and advice throughout the study and in the preparation of the manuscript.

This study was made at the Indiana State Penitentiary with the cooperation of Warden Alfred F. Dowd and the assistance of Drs. P. H. Weeks and P. C. Traver.

A preliminary report was read at the 19th International Physiological Congress, Montreal, Canada, 1953.

Supported by a grant from the Baxter Company, Morton Grove, Ill., and G. D. Searle and Company, Chicago. The department is in part supported by the Michael Reese Research Foundation.

1. Sandweiss, D. J.; Sugarman, M. H.; Podolsky, H. M., and Friedman, M. H. F.: Nocturnal Gastric Secretion, J. A. M. A. **130**: 258 (Feb. 2) 1946.

2. Brown, G. M.; Purchase, E. C. R., and Bresnahan, T. J.: Nocturnal Gastric Contents in Duodenal Ulcer and Non-Ulcer Dyspepsia: Studies of Volume, Acidity and Peptic Activity, Gastroenterology **21**: 331, 1952.

ulcer patients were studied from November, 1951, to May, 1952, and the normal subjects during the same months in 1952 and 1953. Thus, possible seasonal effects on gastric secretion were largely eliminated. The ulcer patients were selected carefully from a group of 156 men with a previous diagnosis of duodenal ulcer on the basis of the following four criteria: (*a*) the presence of typical epigastric distress with nocturnal pain; (*b*) distinct right upper quadrant point tenderness; (*c*) a definite ulcer crater shown by fluoroscopy and x-ray study; and (*d*) no other symptoms or disease.

The normal subjects were selected from a group of volunteers who had been imprisoned for a period of two or more years. Their medical history in the institution was checked, and the drug card was particularly examined to determine whether at any time they obtained antacids, Sippy powders, or alkali for treatment of any type of gastric distress. Any normal subject was rejected for the following reasons: (*a*) history of self-medication of antacids; (*b*) history of hospitalization for gastrointestinal disturbance, and (*c*) the presence of any type of disease. All were given physical examinations and, after the secretion studies, complete gastrointestinal x-ray series were performed. In this group, one patient with silent ulcer and one with prolapse of gastric mucosa were found; they were not included in the normal group.

For the study and work-up, all subjects were hospitalized for a period of at least two days and were fed a soft diet including eggs. Before the secretion tests, food and water were withheld for four and one-half hours. For the nocturnal secretion test, a large Levin tube was passed through the nose and the stomach was emptied for a period of 30 minutes before beginning the collection of the 12 hour secretion. Constant suction was employed by means of a Gomco suction pump. An attendant was assigned to each subject throughout the night for checking the Levin tubes at hourly intervals or more frequently for possible obstruction. The gastric juice was titrated for free and total acidity, and the pepsin content was determined by the method of Bucher, Grossman, and Ivy. A 12 hour urine specimen was collected during the period of nocturnal gastric secretion in 17 ulcer patients and 32 normal subjects, and uropepsin was estimated by a modification of Anson's method. A uropepsin unit was defined as that amount of enzyme that would release 1 mEq. $\times$ 10^{-4} of tyrosine in a 10 minute incubation period at 37 C. The following morning the Levin tubes were disconnected from the constant suction and two 15 minute control period samples were collected for the Hollander insulin test. Then 15 units of insulin were given intravenously, and the samples of gastric juice were collected every 15 minutes for the following two hours. Blood sugar was determined by the method of Nelson.

3. Sandweiss, D. J.; Friedman, M. H. F.; Sugarman, M. H., and Podolsky, H. M.: Nocturnal Gastric Secretion: II. Studies on Normal Subjects and Patients with Duodenal Ulcer, Gastroenterology **7**: 38, 1946.

4. Ivy, A. C.; Grossman, M. I., and Bachrach, W. H.: Peptic Ulcer, New York, Blakiston Company, 1950, p. 696.

RESULTS

All secretion experiments are summarized in table 1 and in the figure. In 34 normal subjects who secreted free acid, the volume of 12 hour nocturnal secretion ranged from 500 to 1,915 ml., with an average of 1,021 ml. The milliequivalents of total free hydrochloric acid secreted during this period varied from 13.5 to 105, with an average of 51. The Hollander insulin test showed a range from 89 to 491 ml. of gastric juice, with an average of 245 ml.; the milliequivalents of free hydrochloric acid ranged from 1.7 to 46.8, with an average of 17.9.

The results of 50 tests performed on 26 ulcer patients showed, in the 12 hour nocturnal secretion, a range from 210 to 2,025 ml., with an average of 945 ml. The milliequivalents of free hydrochloric acid ranged from 6.6 to 131, with an average of 49.6. The two hour Hollander insulin test produced a secretion in the ulcer patients with a range from 53 to 411 ml., with an average of 222 ml. The milliequivalents of free hydrochloric acid ranged from 2 to 32, with an average of 14.8.

Whenever a subject, whether normal or with a duodenal ulcer, showed no free acid in the 12 hour nocturnal secretion or in the insulin test, the test was not included in the averages in table 1. There were three duodenal ulcer patients without free acid in the 12 hour nocturnal secretion, but repeated tests showed a high secretion rate with high acidity, the total milliequivalents of free hydrochloric acid being 48, 56.7, and 29.5. Table 2 shows the results in 10 normal subjects who showed no free acid during a 12 hour nocturnal secretion test. The secretion studies were repeated on four of the subjects and showed values of 29 to 46 mEq. of hydrochloric acid. At this point of the study, it was decided to determine the amylase activity of the gastric contents whenever no free acid was present. Somogyi's amylase method was used, the gastric juice being diluted according to Free and Myers. By this means, we expected to demonstrate neutralization of acidity by regurgitation of duodenal fluids into the stomach. The normal subjects, patients 3 and 4 in table 2, with no free acid, showed 320 and 650 amylase units per milliliter, respectively. On repetition of the tests, free acid was present, but no amylase activity. The remaining six normal subjects showed similar results, namely amylase activity ranging from 440 to 1,530 units per milliliter of gastric juice in the absence of free acidity. We have interpreted this to mean that, in those tests

TABLE 1.—*Summary of All Tests on Normal Subjects and on Duodenal Ulcer Patients*

		Nocturnal Gastric Secretion, 12 Hr.			Hollander Insulin Test		Uropepsin, Units per Hr.
		Volume, Ml.	Total Acid, mEq.	Pepsin Units per Ml.	Volume, Ml.	Total Acid, mEq.	
34 normal subjects	Mean	1,021	51.0	168	245	17.9	206
34 secretion tests	Variation	500-1,915	13.5-105.0	70-290	89-491	1.7-46.8	26-637
26 duodenal ulcer patients	Mean	945	49.6	150	222	14.8	160
50 secretion tests	Variation	210-2,025	6.6-131.0	30-275	53-411	2.0-32.0	23-281

where no free acid was present, a significant amount of regurgitation of duodenal contents occurred, enough to neutralize whatever free acid was secreted. For this reason we have felt justified in not including in table 1 any nocturnal or insulin secretion test that showed no free acid.

TABLE 2.—*Data on Normal Subjects with No Free Acid During Twelve Hour Nocturnal Secretion*

Patient		Volume, Ml.	Free Acid, mEq.	Amylase, Units/Ml.
1	1st test	1,000	0	—
	2nd test	715	46	—
2	1st test	1,025	0	—
	2nd test	725	36	—
3	1st test	725	0	320
	2nd test	770	42	0
4	1st test	735	0	650
	2nd test	925	29	0
5		775	0	440
6		750	0	1,530
7		360	0	600
8		250	0	765
9		935	0	1,335
10		760	0	680

In table 3 the results of the 12 hour nocturnal secretion tests were regrouped according to rate of secretion of total milliequivalents of free hydrochloric acid. In group 1, with free acidity present in all tests, the averages of the normal subjects and of the duodenal ulcer patients were practically identical. However, when all tests, irrespective of presence or absence of free acid, were averaged (group 3) there was a significant difference between normal subjects and duodenal ulcer patients, namely, 39 and 47 mEq. of hydrochloric acid respectively. In group 4, values for those with a high secretion, an output of 60 mEq. and above, showed only a slightly higher average for the ulcer patients as compared to the normal subjects. Yet, in group 6 with low secretion, with 0 to 20 mEq., the duodenal ulcer patients averaged more than twice the milliequivalents of hydrochloric acid than the normal subjects. We could have shown a significant difference also between duodenal ulcer patients and normal subjects, had we compared the results of the high and medium secretion in ulcer patients of group 7 with the

TABLE 3.—*Results of Twelve Hour Nocturnal Secretion Tests Grouped According to Rate of Secretion of Total mEq. of Free Hydrochloric Acid*

Group	Category	Normal Subjects		Duodenal Ulcer Patients	
		No. of Tests	Average mEq. HCl	No. of Tests	Average mEq. HCl
1	Free acid	34	51.0	50	49.6
2	No free acid	10	0	3	0
3	All tests	44	39.0	53	47.0
4	High secretion *	10	75.1	15	85.2
5	Medium secretion †	21	44.0	27	40.6
6	Low secretion ‡	13	4.0	11	9.8
7	High and medium	31	54.0	42	56.0
8	Low and medium	34	28.5	38	32.0

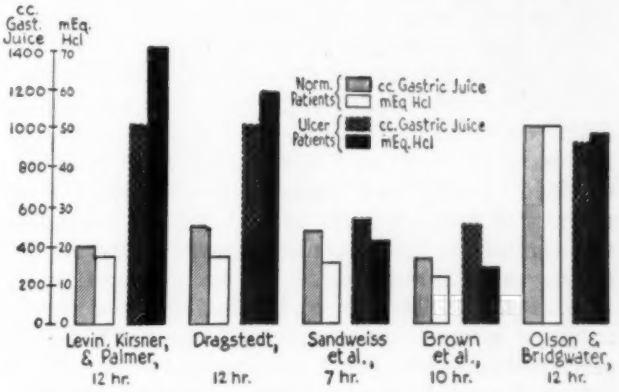
* 60 mEq. and above.
† 20 to 60 mEq.
‡ 0 to 20 mEq.

low and medium normal secretion of group 8, the values for the former being 56 and for the latter 28.5 mEq. of total free hydrochloric acid.

The values for uropepsin and pepsin in normal subjects and in ulcer patients, as determined in 12 hour urine and 12 hour gastric juice respectively, are compared in table 1. In 34 normal subjects, the variation of uropepsin was from 26 to 637 units, with an average of 206 units per hour. The pepsin content of the gastric juice varied from 70 to 290 units, with an average of 168 units per milliliter. In 17 ulcer patients, uropepsin values varied from 23 to 281, with an average of 160 units per hour. The pepsin content of the gastric juice varied from 30 to 275, with an average of 150 units per milliliter.

COMMENT

Comparison of our results with those of other investigators must be limited to those in which continuous aspiration was used for collection of nocturnal secretion. Mears,[5] in 1943, was the first to employ this method, while Chalfen, Winkelstein, Henning and Norpoth, and others have used intermittent aspiration. Mears [5] reported that average volume and acidity were higher in 15 patients with duodenal ulcer than in 50 controls. However, his control subjects, as well as those of a number of other investigators, cannot be considered normal, because they were patients who had either chronic cholecystitis or recurrent appendicitis. In comparing our results with those of other investigators, therefore, we have



Comparison of results of nocturnal secretion studies with those reported by other investigators.

selected only those who have performed the study in a manner similar to our method.

In the figure, the values for nocturnal secretion in normal and ulcer patients, as reported by Brown and co-workers,[2] Sandweiss and associates,[3] Levin, Kirsner, and Palmer,[6] and Dragstedt [7] are compared with our results. Of these five studies, two showed a significant difference between normal and ulcer patients (Levin, Kirsner, and Palmer [6] and Dragstedt [7]). Brown and co-workers [2] and Sandweiss and associates,[3] although they used shorter periods of collection, 7 and 10 hours respectively, did not obtain this marked difference, and our results are in agreement with their findings. Our normal subjects however, showed a higher volume and higher acid content than those of all other investigators. The normal subjects of Levin, Kirsner, and Palmer [6] secreted 18.6 mEq. of hydrochloric acid, those of Dragstedt [7] 18 mEq.,

5. Mears, F. B.: Effect of Atropine on Gastric Secretion During Night, Surgery **13**: 214, 1943.

6. Levin, E.; Kirsner, J. B., and Palmer, W. L.: Continuous Twelve Hour Nocturnal Gastric Secretion in Normal Individuals and in Patients with Duodenal Ulcer After Twenty-Four Hour Fast, Gastroenterology **15**: 454, 1950.

7. Dragstedt, L. R.: Rationale and Technic of Vagotomy, in Sandweiss, D. J., editor: Peptic Ulcer: Clinical Aspects, Diagnosis, and Management, Philadelphia, W. B. Saunders Company, 1951, p. 490.

those of Sandweiss and associates [3] 17 mEq., and those of Brown and co-workers [2] 11 mEq., whereas our normal subjects secreted 51 mEq.

In an attempt to find the reasons for the discrepancy between our results and those of Levin, Kirsner, and Palmer and Dragstedt, the results of our nocturnal secretion tests were regrouped according to rate of secretion of total milliequivalents of free hydrochloric acid, as shown in table 3. Group 1 contains those persons who secreted free hydrochloric acid and it is seen that the difference between normal and duodenal ulcer patients was not significant. However, when all tests, i. e., those with and those without secretion of free acid, were averaged in group 3, the duodenal ulcer patients showed a higher rate of secretion of hydrochloric acid, 47 mEq., as compared to 39 mEq. for the normal persons. The tests were then broken down into groups with high secretion, i. e., secretion of 60 mEq. of hydrochloric acid or above; medium secretion, secretion between 20 and 60 mEq.; and low secretion, secretion between 0 and 20 mEq. Our results would have been similar to those of Sandweiss, recalculated for 12 hour periods, if our normal subjects and duodenal ulcer patients had all had low and medium secretion. In normal subjects, Sandweiss and associates [3] obtained 17 mEq. during a 7 hour nocturnal period, whereas we obtained 28.5 mEq. during a 12 hour nocturnal period in group 8, with low and medium secretion. In duodenal ulcer patients, Sandweiss and associates [3] obtained 22 mEq. for 7 hours, and we obtained 32 mEq. for 12 hours in group 8, with low and medium secretion. On the other hand, we could have shown results in duodenal ulcer patients similar to those of Dragstedt,[7] if our subjects had been grouped as follows. If all our ulcer subjects had had high and medium secretion, group 7, the average of 42 tests would be 56 mEq. of hydrochloric acid, as compared to 60 mEq. in Dragstedt's series. It would appear, therefore, that the type of patient and the experimental procedure employed would greatly influence the amount of nocturnal gastric acid secretion obtained. As table 2 illustrates, one can obtain almost any average if negative secretion tests (no secretion of free acid) for individual patients are included in a series. It was explained above that second tests yielded high amounts of free acid and that the negative tests were due to regurgitation. Our data indicate that regurgitation and neutralization of gastric acid may occur more frequently in normal subjects than in patients with duodenal ulcer.

It is well known that duodenal ulcer patients are persons under considerable tension. In a study of normal subjects for comparison with duodenal ulcer patients, therefore, it would be necessary to obtain the normal persons from the same environment and to perform the tests under the same conditions. The practice of using hospitalized patients as normal subjects cannot give a true comparison. Of equal importance is the question of whether a fair comparison can be made by using medical students or hospital personnel as normal subjects when they are familiar with the tests. We feel strongly that normal subjects should be those persons who are in the same environment, under the same tension and stress as the ulcer patient. It is for this reason that we selected the penitentiary for the study of nocturnal secretion.

There are several possible explanations as to why normal prisoners have a higher level of acid secretion than normal persons on the outside. It can be pointed out that a prison environment is far from normal and that all men are under considerable emotional tension, having a great deal of hostility, resentment, fears, and anxiety, all of which are difficult to eliminate in the abnormal environment of a penitentiary. In other words, these men are living under constant stress and this fact may be reflected in the high uropepsin output in the urine. It has been shown by Spiro and others [8] that administration of corticotropin (ACTH) or cortisone can cause a marked increase in uropepsin output in the urine. Janowitz, Levy, and Hollander [9] reported normal uropepsin values to be 47 units per hour, whereas we obtained an average of 206 units per hour for the normal prisoner, more than four times the reported normal average; on the other hand, our values for duodenal ulcer patients of 180 units per hour of uropepsin are similar to those of 160 units per hour, reported by Janowitz, Levy, and Hollander.[9]

Concerning the peptic activity of the gastric juice, our findings differ from those of Brown and associates.[2] They found twice the value of pepsin secretion in the duodenal ulcer patient as compared to normal controls, whereas we observed the same peptic activity in normal subjects and in duodenal ulcer patients.

It has been demonstrated by Gray and others,[10] Zubiran, Kark, and Dragstedt,[11] and others that corticotropin and cortisone increase the secretion of gastric juice. Therefore, it can be postulated that, as a result of the chronic stress men are exposed to in a penitentiary, there is a marked increase in adrenal cortical activity due to the stimulation by corticotropin from the pituitary. This results in an increased secretion of a number of adrenal steroid hormones including cortisone. Some of these hormones stimulate the stomach to secrete gastric juice and pepsinogen, producing high volumes of acid gastric juice and a high uropepsin output.

A complete survey of the entire population of the prison, 2,400 men, revealed a high incidence of gastrointestinal complaints. This will be reported in a subsequent paper. We mention this because it is to be expected that, when normal men secrete amounts of gastric juice of such high acidity, there would be a considerable number of such symptoms. Having the physiological evidence, namely, that these normal men secrete so much acid, steps should be taken to alleviate the harmful effects of this hypersecretion. The first step would be to be cognizant of the fact that the men complaining of gastrointestinal symptoms may have justification for their complaints and that adequate medical therapy directed toward correcting the symptoms is necessary. The second factor in combating this hypersecretion in inmates of a penitentiary is to consider drastic changes in diet. There are more suggestions that could be entertained, but we

8. Spiro, H. M.; Reifenstein, R. W., and Gray, S. J.: Effect of Adrenocorticotropic Hormone upon Uropepsin Excretion, J. Lab. & Clin. Med. **35**: 899, 1950.

9. Janowitz, H. D.; Levy, M. H., and Hollander, F.: Diagnostic Significance of Urinary Pepsinogen Excretion in Diseases of Upper Gastrointestinal Tract, Am. J. M. Sc. **220**: 679, 1950.

10. Gray, S. J.; Benson, J. A.; Reifenstein, R. W., and Spiro, H. M.: Chronic Stress and Peptic Ulcer, J. A. M. A. **147**: 1529 (Dec. 15) 1951.

11. Zubiran, J. M.; Kark, A. E., and Dragstedt, L. R.: Effect of ACTH on Gastric Secretion in Experimental Animals, Gastroenterology **21**: 276, 1952.

believe that, to rehabilitate a man successfully, he first must be free of symptoms. A person who has a hypersecreting stomach and who has no means of neutralizing the acidity would be less amenable to rehabilitation than one who is symptom-free.

We wish to reemphasize that, whenever a person secretes no free acid for 12 hours, this need not represent the true secretory ability of his stomach. Repeated tests (table 2) have shown that, in seven cases in which no free acid was obtained in the first test, the men were able to secrete large amounts of acid in subsequent tests. The finding of a relatively high amylase content in a gastric juice with no free acid suggests that considerable regurgitation of duodenal contents has neutralized the free acid. Earlier investigators, particularly Winkelstein, found a high incidence of absence of free acid in normal subjects, an incidence of 45%, which is higher than reported by others. The evidence that we present here shows quite definitely that the normal stomach may secrete free acid when tests are repeated. For this reason, whenever we obtained results in nocturnal or insulin secretion studies that showed no free acid, they were not included in the averages. Brown and co-workers [2] suggest, however, that such a procedure does not represent the true character of nocturnal gastric secretion. They state that "gastric contents are the sum of gastric secretion, swallowed saliva and regurgitation of various duodenal contents, and it is misleading to suggest that it is only gastric secretion which is being studied." We agree with this statement, but we want to add that the method of collecting secretion involves many inaccuracies, the greatest of which may be the collection of a combination of duodenal regurgitation and gastric juice. Hence, the greatest source of error in the procedure may occur when gastric acidity is low or absent; on the other hand, when there is high acidity, we can be more certain of ascertaining accurately the true capacity of the stomach to secrete. The fact that we obtained no difference in the two hour Hollander insulin tests between normal and ulcer patients adds further evidence that there is no great difference between the secretory capacity of the stomach of a normal person and of an ulcer patient.

SUMMARY

Twelve hour nocturnal gastric secretion studies showed that normal men in a penitentiary secrete a larger volume and more acid than normal subjects as reported by others. Comparison of the 12 hour night secretion of normal subjects and of duodenal ulcer patients showed no significant difference in volume and acidity, and the gastric secretory response to the Hollander insulin test showed no significant difference between normal and ulcer patients. The uropepsin concentration in a 12 hour urine specimen was higher in normal men in a penitentiary than in normal men reported on by others. However, uropepsin secretion was the same in the normal and in the ulcer group. The peptic activity of the 12 hour nocturnal gastric secretion was the same in both groups of subjects studied.

29th St. and Ellis Ave. (16) (Dr. Olson).

VERTEBRAL FRACTURES AS A COMPLICATION OF ELECTROCONVULSIVE THERAPY

Paul A. Dewald, M.D., Norman M. Margolis, M.D.
and
Herbert Weiner, M.D., Rochester, N. Y.

One of the complications of convulsive seizures, regardless of etiology, is compression fracture of the vertebrae. Lehndorff [1] first reported this occurring in tetanus. Reed and Dancey [2] and Cook and Sands [3] found an incidence of 34.2% and 10.4% respectively in groups of patients with idiopathic epilepsy. After the introduction of pentylenetetrazol (Metrazol) shock treatment, Stalker and Wespi were the first of many to report on the incidence of such fractures with this form of therapy. The reported incidence has varied from 0.375% to 47%.[4] However, in the majority of these papers the number of cases was small, and the design of the studies raised doubts concerning their statistical validity. In the first adequate study of a large series of cases by roentgenograms before and after pentylenetetrazol therapy, Easton and Sommers [4g] reported an incidence of 26.1% fractures in a series of 800 cases. The study included both men and women of ages ranging from 13 to 66 years. The incidence of fractures in men was 37.2%; in women it was 16.8%. On the basis of their study, the authors also concluded that the fifth, fourth, and sixth thoracic vertebrae were most commonly involved, in that order; the incidence of fractures doubled with preexisting osteoporosis; the preexistence of kyphosis, scoliosis, arthritis, nuclear change, or old fracture did not predispose the patient to further fracture; and the age of the patient was not a significant factor.

Most of the studies [5] on the incidence of these fractures in electroconvulsive therapy (ECT) were not adequately controlled, with the result that it is difficult to evaluate the variations of incidence reported. Lingley and Robbins,[6] in a well-regulated study of 230 patients by roentgenograms before and after ECT, reported an over-all incidence of 23% vertebral fractures. The inci-

From the Department of Psychiatry of the University of Rochester School of Medicine and Dentistry and the Strong Memorial and Rochester Municipal hospitals.

Dr. Frederick N. Zuck of the Department of Orthopedics and Dr. George H. Ramsey of the Department of Radiology helped in this study, and statistical analysis was carried out on all results by Dr. Howard Axelrod of the Department of Psychology.

1. Lehndorff, H.: Deformities of Vertebral Column and Ribs in Cases of Severe Tetanus, Wien. med. Wchnschr. **67**: 2477, 1907.

2. Reed, G. E., and Dancey, T. E.: Compression Fractures of Vertebral Bodies Following Induced and "Idiopathic" Convulsions, Canad. M. A. J. **42**: 38, 1940.

3. Cook, L. C., and Sands, D. E.: Spinal Injuries in Convulsive Therapy, J. Ment. Sc. **87**: 230, 1941.

dence in men was 37%; in women, 13.2%. In patients with osteoporosis, the incidence was 43%. The most commonly fractured vertebrae were the fourth and fifth thoracic; younger and older persons were chiefly affected.

When Meschan and co-workers [7] studied 212 male patients receiving ECT, they took preshock roentgenograms, films following each of the first five treatments, and films following the 10th, 15th, 20th, and final treatments. The incidence of vertebral fracture in this group was 35.4%, with the third, fourth, and fifth thoracic vertebrae being most prominently affected. The average number of vertebrae fractured was 2.5 per patient. It was their opinion that the age group 30 to 39 was more liable to fracture than any other age group from 20 to 60 years. There was a high correlation between anterior narrowing of any vertebral bodies on the control film and subsequent fracture. Other factors studied, such as race or presence of spondylosis, kyphosis, or Schmorl's nodes, had no relation to the incidence of fracture. Meschan, Scruggs, and Calhoun did not feel qualified on the basis of their study to make any statement about osteoporosis as a factor in these fractures. In almost two-thirds of the patients, the fractures occurred during the first three treatments and, in four-fifths, within the first five treatments. In 41 cases in which treatment was begun with curare, the incidence of fracture was 19.5%; from 57 cases in which curare was administered after fracture, the authors concluded that the use of the relaxant did not help in preventing further progression of the fracture or the occurrence of a new fracture. Only 13 of 212 patients complained of back pain, and 8 of these had fractures.

DESIGN OF STUDY

Our objective was to provide data regarding the following questions: (1) the incidence of fracture of the spine with and without muscle relaxants; (2) the effects of age, sex, bony structure of the spine, and Glissando and Direct methods of treatment on the fracture incidence; (3) location and severity of fractures; and (4) the incidence of asymptomatic fractures and fractures with minimal transient symptoms. The case material for this study was selected from 1,180 men and women admitted to the two floors for psychiatric patients of the Strong Memorial Hospital and Rochester Municipal Hospital during a 13 month period beginning Jan. 1, 1952. Of these, 263 patients received electroconvulsive therapy. A total of 285 courses of treatment (24.1%) were given to these patients, as 22 of them had 2 courses during the period of study. All of the following data are based on 285 courses of ECT. There were 68 men and 195 women, ranging in age from 14 to 76 years. From 1 to 21 treatments per course of ECT were given, the average number of treatments per patient being 7.5.

Technique.—Before and after each course of treatment a roentgenogram of the lateral dorsolumbar spine was taken. During the course of treatment any complaint of back pain, however minimal, was investigated by additional roentgenograms. In all instances, films were taken by the same technique so that comparison was possible. All patients were treated as they lay supine on a semipadded examination table. The convulsive movements were allowed to follow their own pattern with the exception that light manual restraint was applied to the extremities. A lumbar pad or pad to hyperextend the spine was not used. A rolled gauze mouth gag was used. The electric shock was given by a Med Craft model B-24 machine, which is equipped to deliver the current by the Direct or Glissando methods. With each group the Glissando method was used for six months and the Direct method for the remainder of the study, so that 147 patients were treated by the former and 138 by the latter methods. For various reasons 27 of these Direct method courses and 27 Glissando method courses of treatment were begun and maintained with the muscle relaxant decamethonium bromide (Syncurine). Separate results will be given for the groups treated with and without decamethonium bromide. The dosage of this drug was gaged individually and was considered adequate when there was no resistance to passive stretching during the tonic phase of the convulsion.

Patients Treated Without Decamethonium Bromide.—In the 231 courses of treatment without decamethonium bromide, there were 48 compression fractures of the spine, an over-all incidence of 20.8%. The incidence of fractures in the male population was significantly higher than in the women. Twenty-four out of 56 men, or 42.9%, sustained thoracic fractures, whereas 24 out of 175 women, or 13.7%, sustained such fractures. Although the greatest percentage of fractures occurred in patients under 40 years of age, the number of cases in each age group was so small that this result was not statistically significant (see table).

Direct Versus Glissando: The over-all fracture incidence when the Direct method was used was 25 fractures in 111 courses given, or 22.6%. In 120 courses of treatment given by the Glissando method, there were 23 fractures, or 19.2%. The breakdown of this factor into sexes indicates that, in 13 out of 23 (56.5%) courses of treatment given to men by the Direct method, fractures occurred. In 33 courses of treatment given to men by the Glissando method, 11 fractures (33.3%) occurred. Although not statistically significant, these data would suggest that immediate delivery of a maximal current in men may be conducive to a higher incidence of fracture. In the 88 and 87 women treated by the Direct and Glissando methods respectively, there were 12 fractures in each group, an incidence of 13.6% and 13.8%.

4. (*a*) Cook and Sands.[3] (*b*) von Meduna, L., and Friedman, E.: Convulsive-Irritative Therapy of Psychoses, J. A. M. A. **112**: 501 (Feb. 11) 1939. (*c*) Polatin, P.; Friedman, M. M.; Harris, M. M., and Horwitz, W. A.: Vertebral Fractures Produced by Metrazol-Induced Convulsions in Treatment of Psychiatric Disorders, ibid. **112**: 1684 (April 29) 1939. (*d*) Bennett, B. T., Jr., and Fitzpatrick, C. P.: Fractures of Spine Complicating Metrazol Therapy, ibid. **112**: 2240 (June 3) 1939. (*e*) Hamsa, W. R., and Bennett, A. E.: Traumatic Complications of Convulsive Shock Therapy, ibid. **112**: 2244 (June 3) 1939. (*f*) Krause, G. R., and Langsam, C. L.: Fractures of Vertebrae Following Metrazol Therapy, Radiology **36**: 725, 1941. (*g*) Easton, N. L., and Sommers, J.: Vertebral Fractures in Metrazol Therapy With and Without Use of Curare as Supplement, J. Nerv. & Ment. Dis. **99**: 256, 1944.

5. (*a*) Impastato, D. J., and Almansi, R.: Electrofit in Treatment of Mental Disease, J. Nerv. & Ment. Dis. **96**: 395, 1942. (*b*) Impastato, D. J., and Almansi, R.: Study of Over 2,000 Cases of Electrofit-Treated Patients, New York J. Med. **43**: 2057, 1943. (*c*) Worthing, H. J., and Kalinowsky, L.: Question of Vertebral Fractures in Convulsive Therapy and in Epilepsy, Am. J. Psychiat. **98**: 533, 1942. (*d*) Gralnick, A.: Three-Year Survey of Electroshock Therapy, ibid. **102**: 583, 1946. (*e*) Weinland, W. L.: On Incidence of Fractures in Electro-Shock Treatment, Nervenarzt **22**: 298, 1951.

6. Lingley, J. R., and Robbins, L. L.: Fractures Following Electroshock Therapy, Radiology **48**: 124, 1947.

7. Meschan, I.; Scruggs, J. B., Jr., and Calhoun, J. D.: Convulsive Fractures of Dorsal Spine Following Electric-Shock Therapy, Radiology **54**: 180, 1950.

Effect of Bony Structure: Of 163 courses of treatment given to patients with normal preshock roentgenograms of the spine, there were 34 (20.9%) fractures, whereas in 23 courses given to patients whose initial films indicated any degree of osteoporosis there were 10 (43.5%) fractures. This difference is statistically significant. Of 43 courses of treatment given to patients whose roentgenograms showed degenerative and other changes (such as old fractures, epiphysitis, narrowing of vertebral bodies, kyphoscoliosis, and anterior wedging of bodies), 5 fractures (11.6%) occurred. This difference is not statistically significant when compared with the fracture incidence in patients with normal preshock films.

Location and Severity of Fractures: The number of vertebrae fractured per patient ranged from one to six, with an average of 2.2 per patient. All vertebral fractures occurred in the dorsal area of the spine, with the fifth, fourth, and sixth thoracic vertebrae respectively being most commonly involved to a statistically significant degree. Fractures were considered serious if the degree of compression exceeded by more than one-third the original height of the vertebral body. Of the 49 patients with fractures, there were two such cases. All other fractures consisted of minimal compression of the superior cortical plates of the vertebral bodies.

Treatment During Which Fracture Occurred: In 34 cases the treatment was determined during which the fracture occurred. Twenty-four fractures (70.6%) occurred in the first three treatments while 10 occurred in later treatments. That the majority of fractures occur in the first three treatments is statistically significant.

Symptoms: In the 231 courses of ECT given, 45 patients complained of back pain, and therefore roentgenograms were taken during these courses of ECT. Of these, 34 (75.6%) had fractures. The presence of pain as an indication of fracture is not only statistically significant but is also a strong indication for roentgenographic study. With the exception of two patients with moderately severe persistent pain, all patients had mild and transitory symptoms that consisted of interscapular pain, and, in a few patients, radicular pain corresponding to the level of fracture. However, that fracture may occur without pain is indicated by the fact that 15 (30.6%) of the 49 patients who had fractures were without symptoms, and the fracture was discovered only by routine postshock films.

Other Serious Complications: There were other complications as follows: fracture of neck of humerus, one; avulsion of lesser tuberosity of the humerus, one; dislocation of shoulder, two; dislocation of mandible, two; tear of anterior tibial ligament, one; tear of shoulder capsule, one.

Patients Treated with Decamethonium Bromide.—Fifty-four courses of ECT were begun and maintained with decamethonium bromide. In these, 27 patients were treated by the Glissando method and the rest by the Direct method; there were 8 men and 19 women in each group. Of these 54 patients, 45 were over 50 years old. Analysis of the preshock films in these patients is as follows: negative, 6; osteoporosis, 13; degenerative changes, 14; old fracture, 18; kyphoscoliosis, 3. Of this entire group only one patient (1.9%) sustained a vertebral fracture. This incidence compared with the incidence of fractures in patients who received ECT without a muscle relaxant is highly significant. In 32 of the 34 patients whose fracture was diagnosed during the course of ECT, treatment was continued with decamethonium bromide. Seven of these patients (21.9%) showed either progression of the already existing fracture or new fracture of adjacent vertebrae. Among these patients there were no other serious complications.

COMMENT

The results of our study substantiate the previously known facts that fractures are more likely to occur in men than in women, that age has no statistical value in predicting the occurrence of a fracture, that the fifth, fourth, and sixth dorsal vertebrae respectively are most commonly affected, that most fractures occur during the first three treatments, and that the use of muscle relaxants significantly reduces the incidence of fracture.

Although there has been general agreement concerning these facts, there has been much disagreement on the prevalence of these fractures. We feel it is significant and of interest that in those series similar to ours in which large numbers of patients were studied by roentgenograms taken before and after ECT,[8] there is a high concordance with our results, both as to over-all incidence and as to its variation according to sex difference. This is in sharp contrast to those series reported in which routine x-ray investigations were not carried out. From the above data and the fact that 15 of our 49 fractures caused

Incidence of Fracture During Electroconvulsive Therapy

Age, Yr.	Patients Who Began Without Decamethonium Bromide		Number of Fractures		Percent of Fractures		Patients Who Began With Decamethonium Bromide	
	Males	Females	Males	Females	Males	Females	Males	Females
Glissando								
Under 20	0	0	0	0	....	....	0	0
20-29	2	8	1	2	50	25	1	0
30-39	7	23	3	4	42.8	17.4	1	1
40-49	7	21	3	2	42.8	9.5	0	1
50-59	9	19	2	3	22.2	15.8	0	7
60-69	7	15	2	1	28.6	6.7	5	7
70-above	1	1	0	0	....	....	1	3
Total	33	87	11	12	33.3	13.8	8	19
Direct								
Under 20	0	3	0	1	....	33.3	0	0
20-29	2	15	2	3	100	20	0	0
30-39	3	18	2	2	66.7	11.1	0	3
40-49	4	15	4	1	100	6.7	1	1
50-59	8	25	3	3	37.5	12	2	6
60-69	5	8	2	1	40	12.5	2	6
70-above	1	4	0	1	....	25	3	3
Total	23	88	13	12	56.5	13.6	8	19
Combined								
Under 20	0	3	0	1	....	33.3	0	0
20-29	4	23	3	5	75	21.7	1	0
30-39	10	41	5	6	50	14.6	1	4
40-49	11	36	7	3	63.6	8.3	1	2
50-59	17	44	5	6	29.4	13.6	2	13
60-69	12	23	4	2	33.3	8.7	7	13
70-above	2	5	0	1	....	20	4	6
Total	56	175	24	24	42.9	13.7	16	38

All figures listed as statistically significant showed a chi square validation of at least $P = < 0.02$.

8. Easton and Sommers.[4c] Lingley and Robbins.[6] Meschan.[7]

no symptoms and were discovered only on the film that is taken as routine procedure after ECT, we have concluded that any accurate evaluation of the incidence of these fractures must include films taken both before and after ECT. Clinical facts substantiate this viewpoint because frequently the only thing visible by roentgenogram is minimal compression of the superior plates of the vertebrae, so that, unless a control film is available for comparison, these fractures might be overlooked entirely. The majority of fractures occur early in the course of treatment while the patients are likely to be most inaccessible and when it would be most difficult to elicit the transient pain that accompanies these fractures. This point is illustrated by the fact that Weinland,[5e] in a study of 418 patients using presence of pain as the criterion for x-ray examination, found the incidence of fractures among schizophrenic patients to be one-fifth as high as among depressed patients. He attributes this to the fact that the nature of the schizophrenic illness is such that these patients complain less of physical pain. Analysis of the 15 patients of our series in whom symptoms of fracture were not elicited indicates that 8 of them were severely disturbed and inaccessible at the time ECT was begun. One other patient had a moderate language barrier, and another showed moderate hypochondriasis as part of the presenting illness, so that any back pain may have been masked by the other symptoms. Although pain is not a consistent accompaniment of fracture, its importance is indicated by the fact that 75.6% of our patients who complained of back pain had fractures.

From our series it would seem that the only physical factor that definitely predisposes a patient to fracture is the presence of osteoporosis. Our finding that the incidence of fracture with preexisting osteoporosis is more than double the general incidence confirms the almost identical finding of Easton and Sommers,[4g] who used pentylenetetrazol, and Lingley and Robbins,[6] who used ECT.

Although we cannot statistically support the following hypothesis, it is our impression that a strong muscle mass with violent muscular contraction predisposes to fracture. This is supported by the consistently higher incidence of fractures in men in all series reported. Since the actual mechanism of fracture is still unknown, there have been many attempts to prevent occurrence of fracture by modifications in the technique of administering ECT. These modifications have involved the positioning of the patient, the method of restraint, and the means of initiating the electric shock. Our decision not to hyperextend the spine and to allow freedom of convulsive movements evolved from the feeling that active restraint would predispose to long bone fractures, which are of much greater clinical significance than vertebral fractures. The fact that our incidence of vertebral fractures very closely approximates that of Meschan [7] (in men) and Lingley and Robbins [6] (in men and women), despite the fact that in both these series greater restraint and hyperextension were used, indicates that position of the patient and method of restraint are not significant factors. In regard to the technique of initiating the electric shock, our results comparing the Glissando and Direct methods indicate that this variation produces no appreciable change in the fracture incidence.

From our results it would appear that the only variation in technique that affects the incidence of fractures is the use of a muscle relaxant, as indicated by the fact that the over-all fracture rate fell from 20.8% to 1.9% with decamethonium bromide. The efficacy of this modification in our series contrasts with the 19.5% incidence of fractures found by Meschan [7] with curare. It should be noted, however, that 21.9% of our patients who received decamethonium bromide after sustaining a fracture showed progression of the existing fracture or occurrence of a new fracture. In Meschan's [7] series, 46.6% of his patients who received curare after fracture occurred showed progression of fracture or appearance of a new fracture. These data suggest that muscle relaxants are more effective in the prevention of an initial fracture than in arresting the progression of such fracture or in preventing the appearance of a new fracture.

We are in agreement with previous studies that have indicated that the presence of these fractures is of little clinical significance in the vast majority of cases. The initial symptoms, if present, are minimal and transient and impose little orthopedic disability. Polatin and Linn [9] found no orthopedic or neurological sequelae in their 10 year follow-up of patients sustaining compression fractures during pentylenetetrazol treatment. In none of our cases, including the two "serious" fractures, was orthopedic treatment thought necessary by the consultant.

Despite the relative unimportance of these fractures, it is of obvious advantage to both the patient and the physician to prevent their occurrence when possible. It has been our practice to use a muscle relaxant prophylactically in those patients whose preshock examination shows findings that would predispose to fracture, such as osteoporosis or large muscle mass. Our figures and the data of others [7] indicate that muscle relaxants are not completely effective in preventing the progression or further occurrence of fracture, but as yet there is no statistically reliable series to show what the incidence of this progression would be in continuing treatment after fractures without the use of such relaxants. Until such data are available, we shall continue the use of these relaxants routinely in those patients who have sustained fractures and in whom it was indicated to continue treatments.

SUMMARY

Two hundred and eighty-five consecutive courses of electroconvulsive therapy (ECT) given in a general hospital were studied by means of preshock and postshock roentgenograms to discover the incidence of vertebral fracture. In 72 courses of ECT given to men and 213 courses of ECT given to women, the over-all fracture incidence was 20.8%. The rate in men was 42.9%; in women, 13.7%. Fifty-four patients began ECT with decamethonium bromide (Syncurine) with a resultant fracture incidence of 1.9%. Osteoporosis and sex of the patient were significant in the incidence of fractures. Age, position of patient, restraint, and method of initiating the current did not influence the fracture incidence. The indications for use of muscle relaxants are discussed.

260 Crittendon Blvd. (20) (Dr. Dewald).

9. Polatin, P., and Linn, L.: Orthopedic and Neurological Follow-Up Study of Vertebral Fractures in Shock Therapy, Am. J. Psychiat. **105**: 824, 1949.

CLINICAL NOTES

CARDIAC ARREST AFTER INTRAVENOUS ADMINISTRATION OF PROCAINE AMIDE (PRONESTYL)

REPORT OF A CASE IN WHICH THERE WAS SUBSEQUENT RECOVERY

William Weingarten, M.D.
Nicholas J. Galluzzi, M.D.
and
Alexander A. Doerner, M.D., Staten Island, N. Y.

The quinidine-like properties of procaine on the heart have been known for a long time, but the drug is so rapidly hydrolyzed in the blood stream that the action on the heart is rather unpredictable. Lately procaine amide (Pronestyl) hydrochloride has gained wide use in cardiac arrhythmias. Procaine amide is not hydrolyzed by the body enzymes as quickly as procaine, is therefore longer acting, and has been found to be active when administered orally. Procaine amide can be given intravenously with greater safety than quinidine and on occasion may revert a cardiac arrhythmia to normal sinus rhythm even after quinidine failure.[1] L. C. Mark and co-workers[2] and Goldberg and Cotten [3] have done excellent experimental work on dogs in cardiac arrhythmias and have proved the value of procaine amide in prevention and control of ventricular arrhythmias.

Procaine amide was used successfully on human beings for the treatment of ventricular premature contractions and ventricular arrhythmias. Stearns and co-workers [4] were able to abolish ventricular tachycardia in 7 out of 8 patients, and Kayden and associates [5] reverted ventricular tachycardia in 13 of 15 patients to normal sinus rhythm with procaine amide given intravenously. Mainzer [6] found procaine amide used intravenously to be the drug of choice in the treatment of paroxysmal ventricular tachycardia during anesthesia, particularly for thoracic surgery.

Side-reactions have been noted during the use of this drug and the more common ones are hypotension and widening of the QRS complexes. Severer reactions were chills and fever after oral use of procaine amide,[7] and two cases of agranulocytosis [8] were reported due to the drug. Animal experiments have shown that procaine amide can produce ventricular fibrillation or atrioventricular block and can lead to cardiac arrest. Kayden and associates [5] noted that one of their patients in whom ventricular fibrillation developed died, and Kinsman and associates [9] noted that two of their patients died shortly after administration of procaine amide, but their condition was so poor that no definite conclusions could be drawn as to the relationship of the drug to their demise. Miller and co-workers [10] observed ventricular fibrillation followed by a short period of cardiac arrest when procaine amide was administered to a patient with complete heart block. Acierno and associates [11] noted increase in intraventricular block and subsequent cardiac standstill in one case of ventricular tachycardia treated with procaine amide administered intravenously, but this patient, too, was moribund when treatment was begun, and no definite conclusion could be reached as to causal relationship. On the other hand, the authors noted development of ventricular tachycardia after intravenous injection of procaine amide in a case of auricular flutter and preexisting bundle branch block.

REPORT OF A CASE

In May, 1951, a 30-year-old Negro merchant seaman was found to have pulmonary tuberculosis, and in spite of therapy with streptomycin and *p*-aminosalicylic acid (PAS) a cavity developed in the right upper lobe in January, 1952, and by August, 1952, he needed surgical therapy.

Examination of the cardiovascular system revealed pulse rate of 84 per minute, blood pressure 100/60 mm. Hg, and normal sinus rhythm with no murmurs. The remainder of the physical examination was normal. Resting electrocardiogram was normal. The teleroentgenogram revealed a normal cardiac silhouette.

On Aug. 4, 1952, the patient was subjected to a right upper lobectomy. Preoperative medication consisted of morphine sulfate, 16 mg., scopolamine, 0.65 mg., and secobarbital (Seconal), 100 mg. Endotracheal anesthesia was induced with ether and continued with cyclopropane and oxygen. A slow intravenous drip of procaine hydrochloride (0.6%) was given continuously as a prophylactic measure against cardiac arrythmias. The operation was started at 8:30 a. m.; at 9:00 a. m. 20 mg. of hexamethonium chloride was given intravenously to control the hypertensive level of 210/120 mm. Hg that had developed after intubation. The blood pressure returned to previous levels. An additional 20 mg. was given at 11 a. m. because hypertension recurred. The operation progressed uneventfully, and blood pressure and pulse were stable at 120/70 mm. Hg and 80 per minute respectively until 1:15 p. m. when the pulse was found to be irregular. At 1:25 p. m., an electrocardiogram revealed multiple premature ventricular contractions from the same focus and ventricular tachycardia developed while the tracing was being recorded. At 1:30 p. m., 250 mg. of procaine amide hydrochloride was given slowly intravenously. The continuous electrocardiogram tracing revealed widening of the QRS complexes within one minute (see figure). By 1:40 p. m., the QRS complexes returned to the previous duration, and, since the ventricular tachycardia persisted and the patient's condition seemed precarious, an additional 150 mg. dose of procaine amide was given. At 1:45 p. m., no

From the Department of Medicine, Cardiovascular Research Unit, U. S. Public Health Service.

1. Irvin, C. W., Jr., and Cutts, F. B.: Ventricular Tachycardia: Report of a Case in Which "Pronestyl" Was Effectively Used After Failure with Quinidine, J. A. M. A. **146**: 1282-1283 (Aug. 4) 1951.

2. Mark, L. C.; Kayden, H. J.; Steele, J. M., and Cooper, J. R.: The Physiological Disposition and Cardiac Effects of Procaine Amide, J. Pharmacol. & Exper. Therap. **102**: 5-15 (May) 1951.

3. Goldberg, L. I., and Cotten, M. deV.: Effectiveness of Procaine Amide in Digitalis-Induced Ventricular Tachycardia, Proc. Soc. Exper. Biol. & Med. **77**: 741-744 (Aug.) 1951.

4. Stearns, N. S.; Callahan, E. J., III, and Ellis, L. B.: Value and Hazards of Intravenous Procaine Amide ("Pronestyl") Therapy, J. A. M. A. **148**: 360-364 (Feb. 2) 1952.

5. Kayden, H. J.; Steele, J. M.; Mark, L. C., and Brodie, B. B.: The Use of Procaine Amide in Cardiac Arrhythmias, Circulation **4**: 13-22 (July) 1951.

6. Mainzer, F.: Suppression of Digitalis-Induced Ventricular Premature Beats by a New Procaine Derivative, Lancet **1**: 637-638 (March 17) 1951.

7. Leibowitz, S.: Chills and Fever Following Oral Use of Procaine Amide (Pronestyl), New England J. Med. **245**: 1006 (Dec. 27) 1951.

8. Miller, H.; Pollock, R. C., and Griffith, G. C.: Fatal Agranulocytosis Resulting from a Procaine Derivative, J. Lab. & Clin. Med. **38**: 850-857 (Dec.) 1951. Inouye, M.; Millar, J., and Townsend, J. H.: Agranulocytosis Following Maintenance Dosage of Pronestyl: Report of Severe Case with Recovery, J. A. M. A. **147**: 652-653 (Oct. 13) 1951.

9. Kinsman, J.; Hansen, W. R., and McClendon, R. L.: Procaine Amide (Pronestyl) in the Treatment of Cardiac Arrhythmias, Am. J. M. Sc. **222**: 365-374 (Oct.) 1951.

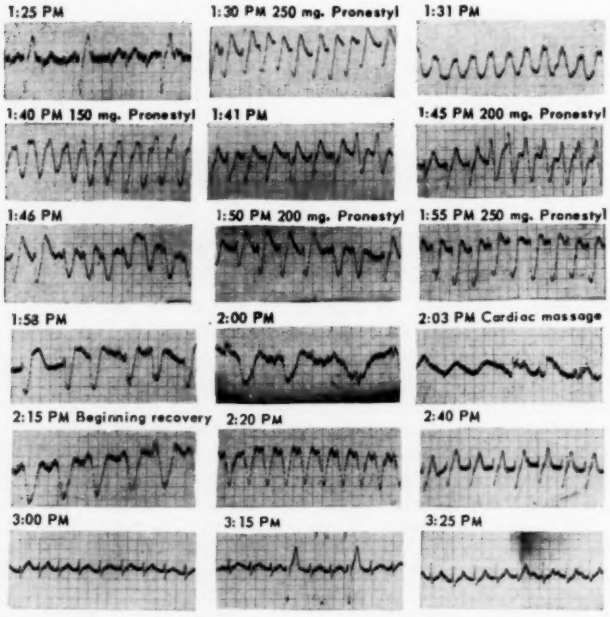
10. Miller, H.; Nathanson, M. H., and Griffith, G. C.: The Action of Procaine Amide in Complete Heart Block, Am. Heart J. **44**: 432-443 (Sept.) 1952.

11. Acierno, L. J.; Gubner, R. D., and Poliakoff, B.: Arrhythmias Produced by Intravenous Procaine Amide, New York J. Med. **53**: 72-75 (Jan. 1) 1953.

change had occurred in the electrocardiogram or in the patient's condition and 200 mg. of procaine amide was given. Within one minute, QRS widening again appeared. By 1:50 p. m., the QRS complexes returned to their original duration and an additional 200 mg. of procaine amide was given, with subsequent widening of the QRS complexes. At 1:55 p. m., although slight QRS widening persisted, in view of the patient's lack of response, an additional 250 mg. of procaine amide was given. During the next few minutes progressive QRS widening occurred, and at 2:00 p. m. cardiac standstill supervened. The exposed heart was immediately massaged manually and epinephrine was injected into the left ventricle. At 2:15 p. m., spontaneous ventricular contractions were observed and the continuous electrocardiogram record demonstrated a reversion to normal in reverse order to the pattern of the evolution of the cardiac standstill. The patient had an uneventful recovery, and several days later a follow-up electrocardiogram revealed an entirely normal pattern.

COMMENT

Although mild and severe reactions during the intravenous administration of procaine amide have been described, to the best of our knowledge no authentic case



Electrocardiogram taken continuously during a right upper lobectomy on a 30-year-old Negro seaman, who had pulmonary tuberculosis.

of cardiac standstill after intravenous administration of this drug has been reported. It is known that during inhalation anesthesia, particularly in intrathoracic explorations, ventricular premature contractions, ventricular tachycardia, and cardiac arrest, may suddenly occur and require emergency treatment. Our patient had received a variety of drugs preoperatively, and during anesthesia, which too may have precipitated the bout of ventricular tachycardia and contributed to the cardiac arrest. In our case there was marked widening of the QRS complexes (which is known to be a side-reaction of procaine amide administration) after each dose of procaine amide, and the progressive widening of the QRS after the last dose led to complete cardiac arrest. In view of the continuous electrocardiogram tracings that were taken during the administration of the drug and revealed immediate changes after each dose, the relationship between the intravenous administration of procaine amide and cardiac arrest seem well established.

ADDENDUM

Since the preparation of this manuscript, a report by Marvin A. Epstein, in the *American Heart Journal* of June, 1953, describes the occurrence of three cases of ventricular standstill during the intravenous procaine amide treatment of ventricular tachycardia.

SURGICAL REPAIR OF INTRAVENTRICULAR SEPTAL DEFECTS

REPORT OF A CASE

Earle B. Kay, M.D.
and
Henry A. Zimmerman, M.D., Cleveland

Evidence reveals that a high proportion of patients with intraventricular septal defects must lead lives of restricted activity and that others die prematurely. Intraventricular septal defects are commonly located high in the septum and connect the left ventricular chamber immediately proximal to the aortic valve to the right ventricular chamber in the region of the median leaflet of the tricuspid valve. Defects in this region may be associated with other defects, such as pulmonic or infundibular stenosis and overriding of the aorta. Less commonly intraventricular defects may be located lower in the muscular septum. They result from failure of the lower ventricular septum growing from the apex to meet and fuse with the downward growing aortic septum.

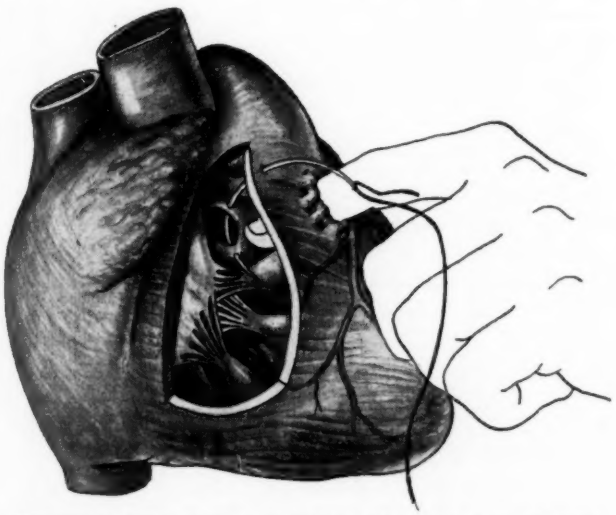
This report deals with the surgical closure of intraventricular septal defects uncomplicated by other abnormalities. The greater pressure within the left ventricle causes a left to right shunt of blood during ventricular systole, increased blood flow to the lungs, pulmonary hypertension, sclerosis of the pulmonary vessels, and cardiac hypertrophy on the right side.

Delay in the surgical treatment of intraventricular septal defects has resulted from the failure of development of an effective but simple heart-lung preparation to allow safe, open operations within the chambers of the ventricles and the danger of air emboli occurring during cardiomyotomy when refrigeration is employed. Closed methods have been employed in which the intraventricular septal defect was plugged with pericardial grafts, but such grafts have been shown to become atrophic after several months.

TECHNIQUE

The technique to be described has the advantages of being simple and safe and adaptable to the closure of the majority of septal defects. An anterolateral exposure through the left third or fourth interspace is employed. The sternochondral junction of either or both the third or fourth costal cartilage is detached from the sternum. Further exposure can be obtained by transecting the sternum or rongeuring the lateral border. A purse-string suture is placed in the anterior wall of the right ventricle in the center of which an opening is made for insertion of the index finger of the left hand to explore the right ventricle, septum, and pulmonary and tricuspid valves for other defects. The size and location of the defect is determined. The jet of blood pouring into the right ventricle

can easily be palpated in the region of the median cusp of the tricuspid valve. By employing a large upholstery needle and no. 1 nonabsorbable surgical suture, the needle is inserted into the ventricular chamber in the region of the pulmonary conus as illustrated in the figure. The tip of the needle is then directed by the index finger within the ventricular chamber to engage the anterior and posterior lips of the septal defect and then through the ventricular wall at a lower level. The needle is removed, and an instrument is passed into the ventricular chamber along the index finger to catch the two ends of the suture and to bring them out of the myotomy opening. The ends are then tied and the knot pushed into the chamber; this approximates the two lips of the defect. Scissors are then inserted along the index finger, and the ends of the suture are cut. It appears that three to four sutures are required to adequately close defects that approach the size of the end of the finger. The myotomy incision is then closed by interrupted suture, and the purse-string suture is removed. The pericardium is closed loosely to allow drainage into



Diagrammatic sketch of the heart showing index finger of the left hand inserted into the right ventricle. The position and character of the septal defect is determined. A large suture ligature is passed through the ventricular wall, through the edges of the septal defect, and out through the ventricular wall.

the pleural space. This is followed by closure of the chest incision in the usual manner with drainage. Refrigeration of the patient adds to the safety of the operation.

REPORT OF A CASE

A 21-year-old white woman was admitted to St. Vincent Charity Hospital on May 20, 1953. For several weeks following birth, she had had a bluish color but otherwise had not been cyanotic. She had had known congenital heart disease since birth. Activity was always restricted because of dyspnea on ordinary exertion. She suffered from frequent respiratory infections and had pneumonia at the age of 13. There had never been any episodes of cardiac failure.

The physical examination revealed a slightly obese woman who was somewhat dyspneic but who otherwise did not appear acutely or chronically ill. The blood pressure was 105 systolic and 70 diastolic. The results of the examination, except for the heart abnormalities, were essentially normal. There was a loud systolic murmur heard over the entire precordium, but this was loudest in the third and fourth interspaces. A thrill was palpable in the same area.

Roentgenograms of the chest, including those taken after a barium swallow, and cardiac fluoroscopy revealed an enlarged right auricle and a prominent pulmonary conus. The heart was not appreciably enlarged. An angiocardiogram revealed a shunt between the right and left sides of the heart with visualization of the aorta within one second. It could not be determined whether the shunt was between the auricles or the ventricles. The aorta appeared small, and the pulmonary conus and artery appeared large. There was delayed passage of the dye through the pulmonary circulation with suggestive evidence of revisualization of the pulmonary artery. The electrocardiogram revealed a right axis deviation. Cardiac catheterization studies gave the following results. Pressure (mm. Hg) values were, in the right auricle, 5/1; in the right ventricle, 80/3; and, in the femoral artery, 118/74. Oxygen concentration (vol. %) was, in the right auricle, 14.2; in the right ventricle, 15.9; in the femoral artery, 17.92; in the superior vena cava, 13; and in the inferior vena cava, 12.5. The oxygen capacity was 18.7 vol. %. The catheter could not be placed in the pulmonary artery on numerous attempts. This is not unusual in patients who have an elevated pulmonary arterial pressure.

An operation was performed on May 27, 1953, through a left anterolateral exposure. The technique employed was similar to that already described. The woman's temperature was lowered to 84 F throughout the operative procedure. The patient withstood the operation well, but the first 48 postoperative hours were complicated by difficulty in maintaining the blood pressure and by pulmonary edema. Thereafter improvement was progressive. The patient was discharged on the 14th day. Check-up examination three months later revealed considerable improvement in the patient's dyspnea.

10465 Carnegie Ave. (Dr. Kay).

CONTRACTION OF LEFT LEAF OF DIAPHRAGM COINCIDENT WITH CARDIAC SYSTOLE

Albert Sjoerdsma, M.D., Ph.D., Bethesda, Md.
and
William B. Gaynor, M.D., Brooklyn

The subject of this report is a patient who showed the phenomenon of contraction of the left leaf of the diaphragm synchronous with systolic movement of the heart. This is the fourth such case to be recorded in the medical literature. The presence of vigorous abdominal pulsations in each of these four patients initially suggested a diagnosis of aneurysm of the aorta. Tetany due to alkalosis or hypocalcemia was found in each instance, and cessation of the abnormal diaphragmatic movements occurred after corrective therapy.

REPORT OF CASE

A 40-year-old white American seaman entered the hospital on the evening of Oct. 6, 1952, with complaints of epigastric pain, nausea, and vomiting of one week's duration. These symptoms had not been relieved by the ingestion of an unknown amount of "baking soda" and had been persistent for two days. He had been aware of a jerking motion of the left upper quadrant of the abdomen for about 24 hours prior to admission. He gave a history of having had yearly episodes of epigastric pain and vomiting for the previous 10 years, each attack lasting one to two days and being relieved by means of a bland diet and "baking soda." The patient had not considered his previous attacks sufficiently incapacitating to warrant medical attention.

Physical Examination.—The pulse rate was 88, respirations 16 (shallow), blood pressure 110/70 mm. Hg, and temperature 97 F orally. The patient was poorly nourished and lethargic,

From the Clinic of General Medicine and Experimental Therapeutics, National Heart Institute, U. S. Public Health Service Clinical Center, Bethesda, Md. (Dr. Sjoerdsma).

Dr. Frederic C. Bartter assisted in the preparation of this paper.

and he showed signs of dehydration. The most striking observation was a heaving motion of the left upper quadrant of the abdomen and the adjacent costal margin, with gurgling noises audible to the examiner. The thrusting motion was coincident with the pulse beat, and the gurgling sound apparently resulted from the movement of air and fluid in the stomach. Auscultation of both lung fields showed a high-pitched hissing sound with each thrust that was distinct from the normal breath sounds. Over the precordium this sound was invariably heard between the first and second heart sounds, resembling

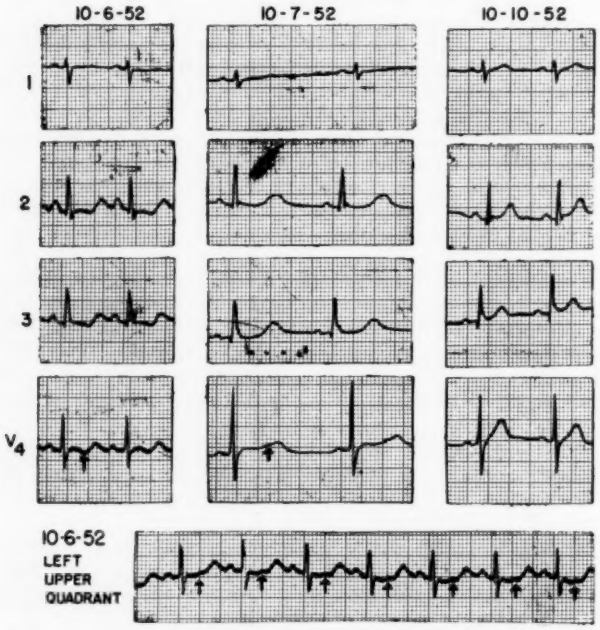


Fig. 1.—Electrocardiograms in patient reported on. On Oct. 6, 1952, the heart rate was 102 and the Q-T interval 0.44 seconds. The prolonged Q-T interval is consistent with the metabolic disturbance. Note the alteration (arrows) of repolarization forces, best seen on lead V-4 and a lead taken from the left upper quadrant of the abdomen in the midclavicular line. This deformity of repolarization forces is interpreted as representing electrical potentials associated with the abnormal diaphragmatic contractions. On Oct. 7, 1952, the heart rate was 56. The prolonged Q-T interval (0.56) persisted. The notching of the T wave on lead V-4 may reflect subthreshold diaphragmatic stimuli, since no abdominal pulsations were noted at the bedside at this time. On Oct. 10, 1952, the heart rate was 88 and the Q-T interval was 0.32. This was a normal tracing. Subsequent records showed no change.

a systolic murmur. Moderate epigastric tenderness was noted, and palpation of the pulsating left upper abdominal region changed the character of the gurgling noises. The Trousseau phenomenon was elicited. The Chvostek sign was not elicited.

Course.—On admission the patient was seen by several examiners, and the cause of the heaving motion was obscure. Aneurysm of the aorta was considered to be a possibility. On the evening of admission, fluoroscopy showed vigorous contractions of the left leaf of the diaphragm coincident with cardiac systole, the contractions being superimposed on normal diaphragmatic movement and having a greater excursion during the expiratory phase of respiration. It was not possible to prevent these contractions by changes in the position of the patient or by full inspiration or expiration. The stomach was distended with air and fluid (during the procedure the patient vomited 250 cc. of a milky-appearing liquid). Electrocardiograms were also done at this time (fig. 1). A spirographic tracing (fig. 2) was obtained by having the patient breathe into a closed-circuit respirometer. The reaction was alkaline to a test of the urine by the use of sodium dinitrophenyl-azo-naphthol disulfonate (Nitrazine). By this time it was apparent that the heaving motions were of diaphragmatic origin. The most likely diagnosis appeared to be chronic duodenal ulcer with metabolic alkalosis and latent tetany.

During the first night of hospitalization 2,000 cc. of 5% dextrose in isotonic sodium chloride solution was given intravenously. Sometime during the night the unusual movement of the left upper quadrant of the abdomen ceased, but the patient continued to be very lethargic and was intermittently disoriented.

The following morning (Oct. 7) roentgenograms of the chest and abdomen were normal; the white blood cell count was 13,000 per cubic millimeter, with a differential count of 72% neutrophils, 18% lymphocytes, and 10% monocytes. The hemoglobin was 13.5 gm. per 100 cc. (Evelyn method) and the hematocrit 45%. The urine gave an alkaline reaction. The specific gravity was 1.019, sugar and acetone negative, and albumin 150 mg. per 100 cc. There were 10 to 15 white blood cells, with many granular and cellular casts per high power field. Serologic studies were negative. Chemical analysis of blood serum revealed sodium, 136.5 mEq. per liter; potassium, 3.12 mEq. per liter; carbon dioxide combining power, 38.2 mEq. per liter; chloride, 70.4 mEq. per liter; pH, 7.79; calcium, 8.6 mg. per 100 cc.; phosphorus, 9.6 mg. per 100 cc., total protein, 5.9 gm. per 100 cc.; and albumin-globulin ratio, 1.4. The blood urea nitrogen was 118 mg. per 100 cc. The data upheld the diagnosis of metabolic alkalosis and indicated renal insufficiency.

During the next three days intensive parenteral fluid therapy was given with solutions containing dextrose, sodium chloride, potassium chloride, and ammonium chloride. By Oct. 10, 1952, the patient's urinary output, which had been low, had returned to normal and the clinical state was much improved. Blood cell counts and blood chemical values had returned to normal except for a blood urea nitrogen level of 87 mg. per 100 cc. From Oct. 10 to Oct. 30, treatment consisted of a progressive Sippy diet and administration of an aluminum hydroxide preparation (Amphogel) and antispasmodics. By Oct. 20 the blood

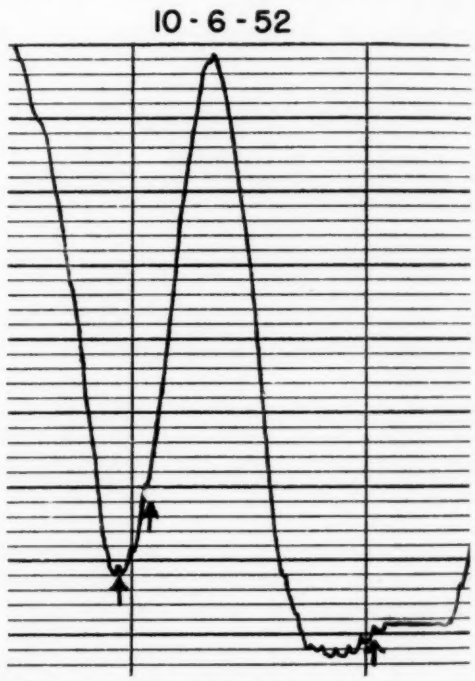


Fig. 2.—Spirogram in patient reported on, taken Oct. 6, 1952. Small upright waves (arrows) represent inspiratory effect of left diaphragmatic contractions. These waves are seen best on maximal expiration. Rate of the diaphragmatic "flutter" measured from the spirographic tracing is about 90 per minute.

urea nitrogen level had fallen to 20 mg. per 100 cc. and urinalysis results were normal. The patient gained 7 lb. (3.2 kg.) in weight, and the night gastric retention, which had been 1,150 cc. on Oct. 10, had decreased to 200 cc. by Oct. 28. Roentgenograms of the upper gastrointestinal tract were interpreted as giving evidence of pyloric obstruction and deformity of the duodenal bulb consistent with chronic duodenal ulcer, although no ulcer crater could be visualized. Gastric analysis after administration of histamine revealed abundant free acid.

A subtotal gastrectomy was performed on Oct. 31, 1952, and the patient made an uneventful postoperative recovery. Examination of the surgical specimen confirmed the diagnosis

of chronic duodenal ulcer. The patient was discharged from the hospital on Nov. 25, fit for light duty. A follow-up visit on Jan. 8, 1953, indicated continued good health with no recurrence of symptoms.

COMMENT

The phenomenon presented in this report has been described in the literature on three previous occasions. The first report was made in 1932 by Karelitz,[1] who described marked pulsation of the left upper quadrant of the abdomen synchronous with the heart beat in a 9-month-old child with rickets and infantile tetany. The possibility of abdominal aneurysm was considered, but fluoroscopy showed that the underlying disturbance was downward movement of the left leaf of the diaphragm. The pulsations disappeared after antirachitic therapy. In 1944, Widström [2] briefly discussed a 37-year-old woman with a probable "gastric" tetany in whom movement of the left upper quadrant of the abdomen and a "splashing" sound from the stomach occurred synchronously with the pulse beat. Again, clonic contractions of the left leaf of the diaphragm were noted at fluoroscopy. No follow-up was reported. Söderström,[3] in 1945, reported the phenomenon in a 24-year-old man with alkalosis and tetany due to vomiting on the basis of pyloric obstruction, secondary to congenital anomalies of the abdominal viscera. Violent pulsations of the abdomen and "splashing" sounds from the stomach simultaneous with the heart beat were at first thought to be due to an enormous dissecting aneurysm. Fluoroscopy later revealed the cause of the disorder to be powerful left diaphragmatic contractions. The abdominal pulsations gradually decreased in strength and finally disappeared, only to recur after fresh periods of vomiting. On several occasions weak contractions of the diaphragm were observed at fluoroscopy when no abdominal pulsations were distinguishable clinically. The patient became symptom free after a resection of the stomach. There is an obvious similarity between these three cases and the one presented in this report. All were diagnostic problems. Whereas all had tetany, this was attributable to hypocalcemia in one and to alkalosis in the others.

Electrocardiograms taken by Söderström [3] showed deformation of the ST and T waves on lead V-4 that were thought to be a manifestation of the diaphragmatic contraction set in motion by the heart. Similar but less marked changes were noted when diaphragm contractions were almost indistinguishable clinically but could still be observed on the fluorescent screen. The changes disappeared when the diaphragm contractions ceased. In 1950, Söderström [4] reported two additional instances of high frequency diaphragmatic activity that was thought to register on the electrocardiogram; this produced a pattern quite distinct from that in his earlier report and in our case. The electrocardiogram (fig. 1) on our patient on Oct. 6, 1952, showed prolongation of electrical systole and depression of repolarization forces (ST-T) consistent with alkalosis and hypokalemia. In addition there was a deformation (note arrows) of repolarization forces that presumably coincided with the diaphragmatic contractions. By Oct. 7, 1952, the heaving motion of the abdomen was no longer evident clinically. However, there was a suggestion of continued electrical activity contributed by the diaphragm appearing as an upward displacement of the ascending limb of the T wave in lead V-4. The spirographic tracing (fig. 2) is the first such recording to be reported on a patient with this disorder. Small inspiratory excursions (note arrows) representing independent activity of the left leaf of the diaphragm were easily recorded during maximal expiration.

What is the mechanism in these persons leading to contraction of the left leaf of the diaphragm with each heart beat? Karelitz [1] offered no explanation. Widström [2] postulated either a direct mechanical irritation of the diaphragm by the heart muscle or a visceromotor reflex with efferent mediation via the phrenic nerve. Söderström [3] ascribed the phenomenon to stimulation of the left phrenic nerve during systole, provoked either mechanically or electrically ("through the heart's own action currents"). Of interest in this regard is a report by Carlson and Jacobson [5] in 1911 directing attention to the frequent occurrence of diaphragmatic contractions synchronous with the heart beat in dogs with severe parathyroid tetany. The contractions were attributed to increased excitability of motor nerves in this condition and were thought to confirm an early observation by Schiff of stimulation of the phrenic nerves by the "action current of the heart." It is clear that the abnormal diaphragmatic stimulation in these various situations is dependent on the increased neuromuscular excitability that exists in the presence of alkalosis and hypocalcemia. Less certain is the manner in which the abnormal impulse is generated. Direct mechanical or electrical stimulation of the diaphragm seems unlikely, since shifts in the relative position of heart and diaphragm in our patient did not prevent the diaphragm from responding with every heart beat. Reflex discharges of cardiac or vascular origin with efferent path via the phrenic nerve probably cannot be ruled out; however, the absence of any case report in which the right leaf of the diaphragm contracted with the heart beat makes this mechanism also appear unlikely. The occurrence of the diaphragmatic stimulation at a time when ventricular volume is decreasing argues against a mechanical stimulation of the left phrenic nerve. The most reasonable hypothesis seems to be that the left phrenic nerve is stimulated by the heart's own action currents. In the human only the left phrenic nerve lies closely adjacent to ventricular myocardium.

SUMMARY

Clonic contractions of the left leaf of the diaphragm synchronous with the heart beat are reported in a 40-year-old man with alkalosis and latent tetany. Three similar cases are cited from the literature. It is postulated that, in the presence of the increased neuromuscular excitability of tetany, electrical stimulation of the left phrenic nerve by the heart's action currents may produce contractions of the left leaf of the diaphragm.

1. Karelitz, S.: Unusual Manifestations of Tetany, Cardiospasm and Unilateral Diaphragm Spasm: Libman Anniversary Volumes, New York, the International Press, 1932, vol. 2, pp. 633-641.

2. Widström, G.: Ett egendomligt fenomenon vid tetani, Nord. med. **23**: 1366-1367, 1944.

3. Söderström, N.: Diaphragm Contractions Synchronizing with Heart Movements, Acta med. scandinav. **122**: 95-102, 1945.

4. Söderström, N.: Clonic Spasm of Diaphragm, Acta med. scandinav. **137**: 27-36, 1950.

5. Carlson, A. J., and Jacobson, C.: Further Studies on Nature of Parathyroid Tetany, Am. J. Physiol. **28**: 133-160, 1911.

MACROSCOPIC DIAGNOSIS IN LIVER BIOPSY

Richard B. Terry, M.D., London

Although the macroscopic features of the specimens obtained by needle biopsy of the liver are often informative and helpful in diagnosis, they have been disregarded largely because the specimens obtained by the usual techniques are unsuitable for naked-eye examination. This report deals (*a*) with a method of obtaining, by biopsy, specimens of the liver of adequate size and in suitable condition for macroscopic examination and (*b*) with the gross appearance of such specimens in various disorders.

The Vim-Silverman needle,[1] which is generally preferred in America, is simple to use. However, the specimen is taken by a split inner needle with a bore of less than 1 mm. that projects only 2 cm. beyond the bevel of the outer needle. The specimen is therefore small, with a maximum volume of about 12.5 cu. mm. It is difficult to prepare for naked-eye examination since it is held inside the split needle, and its gross appearance is distorted by the squeeze of the split needle.

The Iversen-Roholm needle,[2] more popular in Europe, has a bore of 1.6 to 2 mm. and can be inserted into the liver to any desired depth to obtain specimens of 60 to 90 cu. mm. However, the specimen is usually aspirated into the syringe where it often breaks up and is difficult to handle; while a larger specimen is available for histological examination than with the Vim-Silverman technique, it is no more suitable for naked-eye inspection. The same applies to the Franseen [3] needle. The disadvantages of the Iversen-Roholm method may be overcome without losing its advantages by combining the syringe, trocar, and needle into one instrument with suitable modifications; this instrument,[4] hereafter referred to as the combined instrument, gives large intact specimens that are easily handled and readily examined by the naked eye.

METHOD

The Combined Instrument and Its Use.—The instrument (fig. 1) consists basically of a syringe carrying a biopsy needle on the barrel and a trocar on the plunger. A spring catch on the cap of the syringe engages with the notched piston rod and permits negative pressure to be maintained when the plunger is retracted. In taking the biopsy specimen, the left hand grips the piston handle keeping the plunger, and thus the trocar, stationary, while the right hand thrusts the barrel forward, thus introducing the needle into the liver. In this single movement, the specimen is cut out by the needle, the trocar is removed from the distal end of the needle, negative pressure is produced within the syringe, and the plunger is locked in its retracted position maintaining suction on the biopsy specimen without further attention from the operator. After rotation of the needle to and fro to loosen the base of the core, the needle is withdrawn.

Manipulation of Biopsy Specimen.—The core of liver is placed in a glass tube, 5 cm. long with a 4 mm. bore, where it can be freely handled without damage and its gross appearance studied with both the naked eye and a hand lens. The glass tube magnifies the core somewhat and permits transillumination; it also keeps the core straight and intact, facilitating histological preparation.

Fig. 1.—The combined liver biopsy instrument.

To introduce the specimen into the glass tube, the needle is unscrewed from the syringe and a blunt trocar of appropriate size inserted into the needle until the specimen just appears at the bevel; the point of the needle is then placed well inside the glass tube, and the specimen is gently deposited in the tube by retracting the needle with the trocar held still. Any blood on the specimen is immediately washed away and aspirated from the glass tube; the syringe and procaine already employed for local anesthesia may be used for this purpose. The specimen must be examined without delay and before it is placed in fixative, which would make it appear homogenous.

GROSS APPEARANCE

Macroscopic Diagnosis.—The specimen of normal liver has a milk chocolate hue, which varies in depth with the concentration of glycogen and which on closer inspection is seen to be slightly mottled, the darker areas corresponding to the central zones. The specimen is opaque to transillumination, and no vascularity is visible within the tissue.

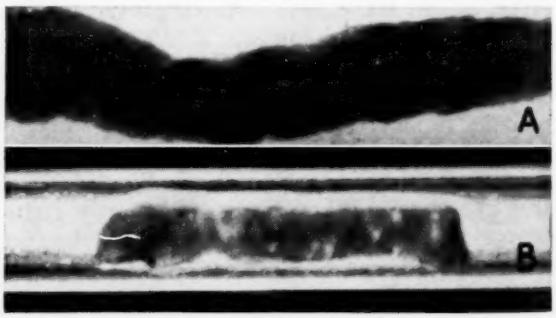


Fig. 2.—*A*, speckled biopsy specimen showing obstructive jaundice; *B*, transilluminated biopsy specimen showing translucent follicles of Boeck's sarcoid.

In obstructive jaundice, the specimen has a speckled appearance (fig. 2*A*); against a background paler than normal, the central zones appear as dark green irregular spots up to 1 mm. in diameter. This picture has been noted in each of 27 specimens from patients with obstructive jaundice. If in a patient with jaundice this characteristic picture is absent, the jaundice is probably not

From the Hektoen Institute for Medical Research of Cook County Hospital and the departments of pathology of Cook County Hospital and Northwestern University Medical School, Chicago; now at St. Bartholomew's Hospital and the Seamen's Hospital, London. Fellow of the Dr. Jerome D. Solomon Memorial Research Foundation.

This study was supported by a grant from the Dr. Jerome D. Solomon Memorial Research Foundation.

1. Silverman, I.: New Biopsy Needle, Am. J. Surg. **40**: 671, 1938.

2. Iversen, P., and Roholm, K.: On Aspiration Biopsy of Liver, with Remarks on Its Diagnostic Significance, Acta med. scandinav. **102**: 1, 1939.

3. Franseen, C. C.: Aspiration Biopsy, with Description of New Type of Needle, New England J. Med. **224**: 1054, 1941.

4. Gillman, T., and Gillman, J.: Modified Liver Aspiration Biopsy Apparatus and Technique, with Special Reference to Its Clinical Applications as Assessed by 500 Biopsies, South African J. M. Sc. **10**: 53, 1945. Terry, R.: Needle Biopsy of Liver, with Special Reference to Modified Gillman Technique, Brit. M. J. **1**: 657, 1949.

due to obstruction. This green speckling is, however, not pathognomonic of extrahepatic obstruction, since it is occasionally observed in hepatitis with marked biliary stasis, especially the cholangiolitic type. It is not only the macroscopic appearance that is deceptive, for it is in these very cases that the histological picture often fails to differentiate between parenchymal and obstructive jaundice. The green speckling is not destroyed by fixation, but is rather accentuated since the fixative bleaches the rest of the lobule.

In viral and toxic hepatitis and in jaundice complicating cirrhosis, the specimen is pale and uniformly icteric, without any green speckling. If some speckling is present, it is usually slight and, apart from cholangiolitic hepatitis, never approaches the speckling of obstructive jaundice, either in size of spot or depth of green. In the acute stage of viral hepatitis, hyperemia is sometimes clearly visible.

In fatty metamorphosis the whole core is rather pale and is moderately translucent on transillumination. The specimen also appears anemic. In cirrhosis the core is also pale but is not translucent unless much fat is present. In two instances of advanced postnecrotic cirrhosis with the diagnosis of grossly nodular livers confirmed at necropsy or at operation, the specimens disintegrated in spite of careful handling; similarly, numerous specimens obtained by the biopsy needle at necropsy also disintegrated despite every precaution. Careful microscopic examination of the needle specimens obtained both from the patients and from the cadavers revealed no evidence of cirrhosis. Histological examination of large slices of liver at postmortem examinations revealed many lines of cleavage between the parenchyma and the coarse fibrous bands, and it appeared that the biopsy specimens must have fragmented at similar weak points. Thus fragmentation of the biopsy specimen despite careful handling should suggest the possibility of hepatic cirrhosis, even without histological confirmation.

Metastatic carcinoma of the liver has a dead-white appearance and is most obvious when the metastasis stands out in marked contrast in a specimen that is mainly normal. When the whole core is neoplastic it is still quite obvious, but the pallor of a cirrhotic specimen may mislead one for want of a standard; if one is in doubt, it is unlikely that the appearance is due to metastasis. On four occasions creamy pus-like fluid has been aspirated; smears revealed necrotic malignant cells.

The follicles of Boeck's sarcoid and of miliary tuberculosis are not apparent on superficial examination. However, if the specimen is transilluminated, the rounded follicles are easily detected, for they are brightly translucent and contrast sharply with the opaque normal tissue (fig. 2*B*). This translucency has to be looked for in the fresh specimen, as it dims gradually at room temperature and disappears immediately on fixation. Once the follicles have been localized by transillumination, they are easily detectable under direct light as well-defined pale areas. These appearances have been noted in each of the 11 cases of sarcoidosis and 2 of miliary tuberculosis in which biopsies have been done since this observation was first made. The pathologist is frequently unable to differentiate the various granulomas unless he can find caseation or tubercle bacilli. Therefore the gross demonstration of the translucent foci, the granulomas, may be just as helpful as the microscopic examination for correlation with the clinical findings. Detection of the follicles also indicates at once that the biopsy has been successful; if these foci are not seen, the biopsy may be repeated without waiting for the histological report, especially if the clinical evidence for some granulomatous condition is strong. Finally the position of the follicles may be reported to the pathologist, thus allowing him to cut his sections in the right place.

Pallor and translucency should be carefully evaluated when present at the proximal end of the specimen, where they are often seen as a result of the suction applied during the biopsy; although they may mislead the unwary, they do not approach the white opacity of carcinoma or the clear translucency of the granulomas. The glass tubes should therefore be marked at one end with a substance such as sealing wax and the specimen should always be inserted through that end.

In hemochromatosis, the biopsy specimen is at once seen to be unusually brown in color; after the specimen has been standing in methylal-saline solution this hue intensifies, in contrast to other specimens, which are blanched by this fixative. Furthermore, the pale strands of fibrous tissue stand out against the brown color, permitting one to make an accurate forecast of cirrhosis in addition to hemochromatosis.

OBSERVATIONS

Observations During Liver Biopsy.—The duration of bleeding from the skin incision is an excellent indication of adequate hemostasis; thus the biopsy should not be performed until the incision has stopped bleeding. If the bleeding lasts longer than 10 minutes, the biopsy should be postponed for further investigation. This precaution has saved me from performing biopsies on patients with abnormal bleeding tendencies due to uremia, thrombocytopenia, and low prothrombin levels, which were incorrectly reported as normal or which fell suddenly from normal.

The resistance to the needle is increased in well-developed cirrhosis, but not in metastatic carcinoma. Patients with hepatic metastases usually experience no sensation of any kind during the biopsy; since other patients are usually aware of a vague sensation of pushing or pulling, it seems probable that the nerve supply of Glisson's capsule is often interrupted in metastatic carcinoma of the liver.

Either during infiltration with a local anesthetic or during the actual biopsy, the presence of ascites may be determined by withdrawing ascitic fluid into the syringe or, on occasion, by collecting some fluid escaping through the biopsy track. Similarly one may obtain clear fluid in cystic disease of the liver, pus from an amebic abscess, blood from a hemangioma, bile from a distended bile duct in obstructive jaundice, and hooklets from a hydatid cyst.

Application to Other Techniques.—The specimen obtained with the Vim-Silverman needle lies rather obstinately within the split inner needle, from which it is usually dislodged with some form of hook and delivered straight into the fixative. To permit naked-eye examination, the biopsy specimen should be removed with a piece of nonglossy paper, to which it readily adheres, or with a

coverslip, which facilitates transillumination. With the Iversen-Roholm, Franseen, and similar aspiration biopsy techniques, the specimen is usually sucked into the syringe. If the syringe is partly filled with saline solution before suction is applied, the specimen is less likely to be damaged and is more easily handled and inspected.

COMMENT

While it is clearly advantageous to be able to determine at the bedside whether a liver biopsy specimen is abnormal and what the abnormality is, it is doubtful whether this advantage alone would recommend the combined instrument in preference to either the Vim-Silverman or the Iversen-Roholm technique. It is therefore emphasized that the combined instrument has other advantages, including simplicity and rapidity of action.

The mortality rate does not preclude larger biopsy specimens, for the rate is about 0.12% with either the Vim-Silverman or the other techniques.[5] It is probable that in the Vim-Silverman technique the size of the outer needle is more important than the small size of the biopsy specimen in the production of complications and that the double action of the two needles damages the liver more than the single needle of other methods. Using the combined instrument with needles of 2.1, 1.8, and 1.65 mm. internal diameter, I have performed over 300 biopsies without hemorrhage or death and with but one major complication, which was biliary peritonitis in a patient with intrahepatic biliary obstruction.

SUMMARY

A method of obtaining liver biopsy specimens of adequate size and suitable condition for naked-eye examination is reported. The characteristic macroscopic appearances are described; such conditions as parenchymal jaundice, obstructive jaundice, metastatic carcinoma, and granulomas, including tuberculosis and Boeck's sarcoid, may be readily recognized at the bedside.

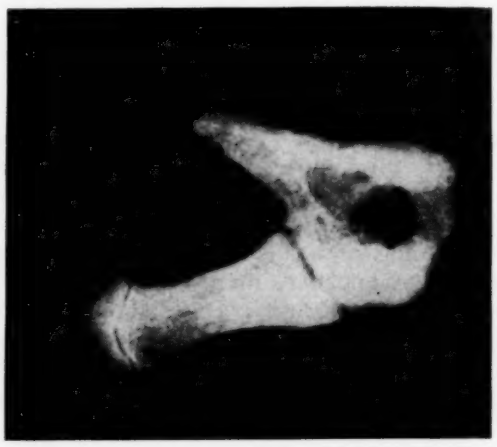
5. Terry, R.: Risks of Needle Biopsy of Liver, Brit. M. J. 1: 1102, 1952.

DEFORMITY OF THE XIPHOID CARTILAGE ASSOCIATED WITH GASTRO-INTESTINAL SYMPTOMS

C. R. Hanlon, M.D.,† Bethlehem, Pa.
and
Mary Margaret Miller, M.D., New York

Scant attention has been paid to lesions and abnormalities of the xiphoid cartilage causing gastrointestinal symptoms. This is probably so because the xiphoid is seldom injured, and congenital or developmental abnormalities are seldom noted or looked for. The xiphoid process, the inferior portion of the sternum, juts down into the anterior abdominal wall in front of the liver and can be palpated in the floor of the depression known as the epigastric fossa, or the "pit of the stomach." The process is usually flat and pointed but may be almost any shape. It consists of cartilage with a core of bone that enlarges with age. Trauma to the xiphoid is observed infrequently since the cartilage is sheltered in the subcostal angle and is usually more posterior than the body of the sternum; however, it may be contused or broken by a force applied against the anterior wall of the chest.

The epigastric syndrome[1] is the term used to refer to epigastric pain localized in the xiphoid region, arising from a malformed but unbroken xiphoid process. The xiphoid is tender on palpation. The patient complains of heaviness in the pit of the stomach. Nausea and vomiting are noted only when the xiphoid is bent back, and there may be pain on deep breathing. Certain actions, such as eating, applying pressure over the xiphoid, and sitting up, increase the discomfort. Lying in the supine position is stated to lessen the pain. We have encountered a case of tack hammer deformity of the xiphoid associated with gastrointestinal symptoms. This is the second such case reported in the literature as far as we have been able to determine.



Roentgenogram of surgically removed xiphoid cartilage of the patient in case reported on, showing tack hammer deformity.

REPORT OF A CASE

A 46-year-old white man was admitted to St. Luke's Hospital on May 12, 1952, complaining of epigastric tenderness of three weeks' duration. There was a history of slight trauma to the xiphoid process when the patient was struck by a baseball as a child. In 1937, the patient had had gnawing, crampy, epigastric pain, and he was put on an ulcer regimen, which did not relieve the symptoms. The pain continued intermittently, occasionally relieved by medication. There was a constant bitter taste in the mouth and frequent gaseous bloating and substernal burning following ingestion of food. There was occasional nausea accompanying the substernal burning, with relief afforded by belching or vomiting. There was no intolerance to fatty food. There was no relief by epigastric massage. When the patient was reclining, his symptoms were eased by lying on the left side. There was no pain related to respiration. Despite symptoms, the patient's appetite remained good, and there was no weight loss. For three weeks before admission, the patient noted exquisite pain in the epigastrium overlying the xiphoid process when pressure was exerted in this area.

Examination revealed a subcutaneous anterior protrusion of the xiphoid with a fishtail deformity of the inferior aspect of the cartilage. The larger prong of the xiphoid was on the right. Movement of the xiphoid reproduced the exquisite pain of which the patient complained. There was mild tenderness to deep palpations in the right upper quadrant of the abdomen and in the epigastrium.

† Dr. Hanlon died on Oct. 18, 1953.
Resident at the Memorial Center for Cancer and Allied Diseases, New York (Dr. Miller).

1. Burman, M., and Sinberg, S. E.: Injury of the Xiphoid, New York, Columbia University Press, 1952.

Laboratory studies, including a complete blood cell count, urinalysis, and serologic studies, were all normal. Unfortunately, lateral and oblique roentgenograms were not secured before operation. Excision of the xiphoid was performed. A lateral transverse incision was made over the xiphoid process and extended down to the bone. The muscular and fascial attachments of the process were excised subperiosteally, exposing a tack hammer type of xiphoid (see figure). The deformed cartilage was then excised at its articulating surface with the sternum. Closure was routine, wire being used in the skin. The xiphoid measured 5 by 2 by 0.5 cm., with the right lower prong 1.9 by 1.6 cm. and a left prong of 0.3 by 1 cm.

The patient was discharged from the hospital on the third postoperative day. Wound healing was by primary intention. One year after the operation, the patient reported no recurrence of symptoms and considered himself cured.

EPITHELIOMA AND FIBROSARCOMA OF THE PENIS OCCURRING SIMULTANEOUSLY

James H. McDonald, M.D.
and
Norris J. Heckel, M.D., Chicago

The simultaneous combination of fibrosarcoma and epithelial carcinoma in the same penile lesion has not been reported previously. Because of this the following case history is of interest.

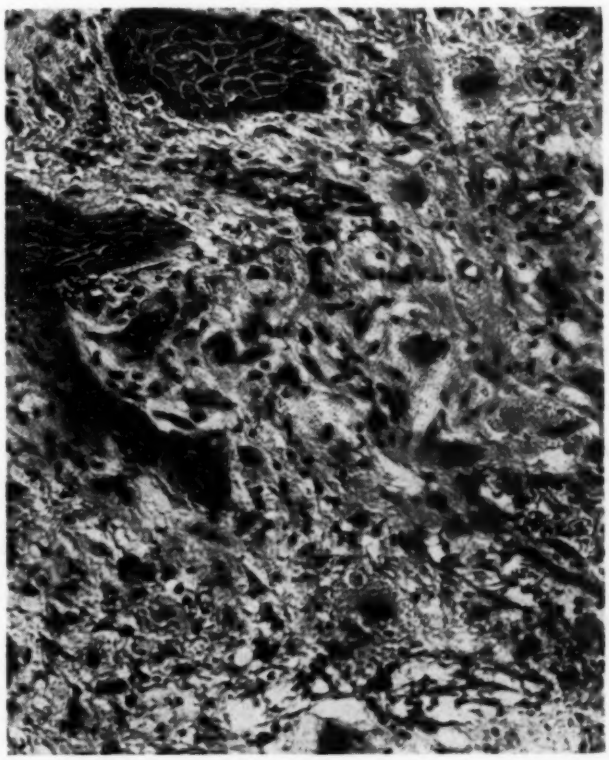
REPORT OF A CASE

A 70-year-old white man was admitted to the Presbyterian Hospital on May 17, 1951, with the complaints of a swelling of the glans penis, which he had noticed two weeks earlier, and hemorrhage on two occasions from beneath the prepuce. For the past 30 years, the patient could not retract the prepuce.

The patient was moderately obese and did not appear ill. Examination of the external genitalia revealed a stony hard mass 2 cm. in diameter on the left dorsal aspect of the glans penis beneath the prepuce. A dark brown serous secretion oozed from beneath the prepuce. There were no palpable inguinal lymph nodes. The testes were slightly atrophic. The prostate was normal per rectum. The remainder of the physical examination was normal. On admission the patient's hemoglobin was 13.5 gm. per 100 cc., the red blood cell count 4,480,000, and the white blood cell count 5,500. Examination of the urine revealed 3 red blood cells and 10 white blood cells per cubic millimeter. Culture of the urine revealed no growth.

On May 22, 1951, with the patient under general anesthesia, a dorsal slit was performed, and a well-circumscribed tumor about 2 cm. in diameter was exposed on the left dorsal aspect of the glans penis. A frozen section biopsy revealed a squamous cell carcinoma. Partial amputation of the penis was carried out. The postoperative course was uneventful, and the patient was discharged from the hospital 21 days after the operation.

The microscopic examination revealed a well-differentiated squamous cell carcinoma arising from an ulcerating epidermis. There were frequent whorling and pearl formation. Scattered through the underlying stroma were numerous tumor giant cells originating from the stromal cells. Transition between the proliferating fibroblasts and the bizarre giant sarcoma cells was observed. The giant cells contained single and multiple nuclei that were highly irregular in shape and staining qualities (see figure). The diagnosis was (1) mixed malignant neoplasm of the penis, (2) squamous cell carcinoma of the penis, and (3) fibrosarcoma of the penis. The patient was followed for 27 months and was completely well. There was no evidence of local or distant metastases.



Photomicrograph showing bizarre giant sarcoma cells and proliferating fibroblasts (× 950).

COMMENT

Of the 12 cases of fibrosarcoma of the penis reviewed in the literature, two were associated epithelial carcinoma.[1] Wheelock and Clark [2] reported both neoplasms in a patient but in separate penile lesions. Warren and Sommer [1b] found a fibrosarcoma in the stroma of an epithelioma of the penis after five and one-half years of unsuccessful treatment of the carcinoma.

The metastatic pattern of the fibrosarcoma is of importance in delineating the desirable operative procedure. Metastases disseminate primarily by the blood stream and are generally believed to occur late in the disease. The lungs are commonly involved in metastases, and the regional lymph nodes are not usually affected.[3] In these lesions, without local lymph node metastases, partial penile amputation is the procedure of choice.

122 S. Michigan Ave. (3) (Dr. Heckel).

From the Department of Urology, Presbyterian Hospital, and the University of Illinois College of Medicine.

1. (*a*) Wattenberg, C. A.: Primary Fibrosarcoma of Penis: Review of Literature and Report of Case, J. Urol. **51**: 543-547 (May) 1944. (*b*) Warren, S., and Sommer, G. N. J., Jr.: Fibrosarcoma of Soft Parts, with Special Reference to Recurrence and Metastasis, Arch. Surg. **33**: 425-450 (Sept.) 1936. (*c*) Leveuf, J.: Soc. de Pediat. de Paris Seance **2**: 209, 1944-1945. (*d*) Hidal, M. T., and Sadi, Al: Tumores do pênis: Consideracões a propósito de um caso de sarcoma fibroblástico, Rev. Paulista Med. **36**: 101-110 (Feb.) 1950. (*e*) Fuentes Ferrer, J. J.: Arch. Hosp. univ. Garcia, Habana **2**: 335-340 (May-June) 1950.

2. Wheelock, M. C., and Clark, P. J.: Sarcoma of Penis, J. Urol. **49**: 478-481 (March) 1943.

3. Heller, E. L., and Sieber, W. K.: Fibrosarcoma: Clinical and Pathological Study of 60 Cases, Surgery **27**: 539-545 (April) 1950. Wilson, D. A.: Tumors of Subcutaneous Tissue and Fascia, with Special Reference to Fibrosarcoma—Clinical Study, Surg., Gynec. & Obst. **80**: 500-508 (May) 1945.

DIFFICULTIES IN EARLY DIAGNOSIS OF MYOSITIS OSSIFICANS

Leonard Weinstein, M.D.
Samuel H. Fraerman, M.D.
and
Philip Lewin, M.D., Chicago

Myositis ossificans is a benign condition in which there is degeneration of muscle and connective tissue, followed by replacement of these structures with a cellular fibrous tissue that eventually becomes ossified.[1] The usual case of myositis ossificans circumscripta has a definite history of injury to the involved part, followed by pain and swelling in that area. Finally, after several weeks, the typical cloud-like formation of calcified tissue is seen in the muscle mass adjacent to the bone.[2] Ordinarily there is little difficulty in diagnosing this condition. In the atypical case, however, the diagnosis may become difficult. This case was such a problem. Because of this patient's unusual history and symptoms and because of the problems involved in the differential diagnosis, we feel justified in reporting this case.

REPORT OF A CASE

An 8-year-old boy was brought to our office March 10, 1951, for routine orthopedic examination. He was under treatment for flatfoot and came to be fitted for a corrective shoe. His mother stated that for the past few days he had had some pain in his left thigh and that he limped. Examination showed nothing in the left hip, knee, or foot to account for the symptoms. The patient was advised to rest. On reexamination four days later, there was pain, swelling, and tenderness over the anterior aspect of the left thigh. A roentgenogram showed soft tissue swelling and a faint periosteal elevation of the middle third of the femoral shaft. The child was hospitalized at once for diagnosis and treatment. At this time the patient and his family were asked whether any kind of trauma had occurred. They could not remember any specific injury. Although no history of trauma could be obtained from the patient or his family, the mother stated that on March 3 or 4 the family had been in the country. During this holiday the patient had climbed hills and had gone tobogganing, but he denied injuring himself. Shortly after their return home, he began to complain of pain in his left thigh. The pain was described as an ache aggravated by movement, causing him to limp. The history was noncontributory. The child had had measles, rubella, mumps, and chickenpox. He had had no recent upper respiratory infection but had an allergy to dust for which he was taking 25 mg. of tripelennamine (Pyribenzamine) hydrochloride twice daily.

Physical examination on March 14, 1951, the day of admission to the hospital, showed a young boy ambulatory but limping because of pain. The left leg was held in external rotation at the hip and in flexion at the knee. Motions to straighten the leg were possible but painful. There was moderate fusiform swelling of the anterior aspect of the left thigh, with diffuse tenderness throughout this area. There was no evidence of cellulitis, nor was there any lymphadenopathy in the groin. His temperature on admission was 101.4 F. Physical examination otherwise showed nothing significant. The patient was given penicillin by injection and placed on a regimen of bed rest.

From the Orthopedic Department, Michael Reese Hospital.

1. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 3, Philadelphia, J. B. Lippincott Company, 1949.

2. Bowers, R. F.: Myositis Ossificans Traumatica, J. Bone & Joint Surg. **19**: 215, 1937. Mercer, W.: Orthopaedic Surgery, ed. 4, Baltimore, Williams & Wilkins Company, 1950. Painter, C. F.: A Consideration of the Etiological Factors in Myositis Ossificans Traumatica, Boston M. & S. J. **184**: 45, 1921.

The patient's hemogram showed a hemoglobin level of 11.8 gm. per 100 cc., or 76% (photoelectric colorimeter, with 15.6 gm. per 100 cc. as 100%); a red blood cell count of 3,810,000 per cubic millimeter; a white blood cell count of 8,350 with a differential count of 47% segmented polymorphonuclear leukocytes, 3% band cells, 4% eosinophils, 1% basophils, 39% lymphocytes, and 6% monocytes. The serum calcium level was 11.1 mg. per 100 cc., and the serum phosphorus level was 5.9 mg. per 100 cc. The serum alkaline phosphatase was 9.2 units, the normal value being between 3 and 15 units. The total serum proteins were 7.1 gm., with the albumin 4.6 gm.

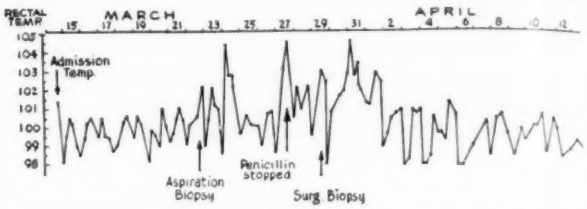


Fig. 1.—Temperature chart of patient in case reported on from time of admission until discharge from hospital.

and globulin 2.5 gm. Agglutination tests for typhoid, paratyphoid, and dysentery organisms were negative. The Kahn test was negative, and the urine was normal. The chest film was normal, and the patch test was negative for tuberculosis. Follow-up white blood cell counts with their differential cell counts were also within normal limits. The temperature graph was that of a septic process (fig. 1).

On March 20, 1951, six days after the patient's admission and about two weeks after the apparent onset of the symptoms, the swelling of the thigh had increased considerably. Induration was present, and the veins of the skin were enlarged and prominent. These observations plus the roentgenogram showing periosteal reaction in the face of a normal white blood cell count caused us to consider bone sarcoma as a possible diagnosis. Since there was some question as to the exact diagnosis, however, other opinions were obtained. These impressions were divided between bone tumor and infection (fig. 2*A*).

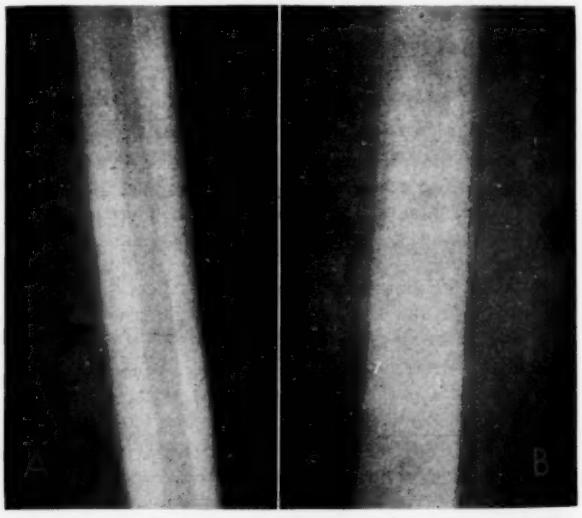


Fig. 2.—Roentgenograms of patient in case reported on. *A*, on a film taken March 21, 1951, definite periosteal reaction is visible. *B*, On a film taken April 6 the typical cloud-like formation of ossifying tissue around the femoral shaft is seen.

An aspiration biopsy was done. Fluid withdrawn from the knee was clear and sterile on culture. From the area of maximum tenderness, flecks of clotted blood and amorphous material were obtained. From this material a nonhemolytic Staphylococcus culture was obtained. The Staphylococcus later proved to be a contaminant. After the biopsy the temperature rose several degrees, with peaks of 104 F. The leukocyte count was still within normal limits. Penicillin therapy was stopped.

On March 28, 1951, because of the continuing process and because no definite diagnosis had been made, a surgical biopsy

was done by an anterior approach. As soon as the skin was incised, generalized edema of the subcutaneous tissue was seen. When the deep fascia of the thigh was opened, gray-pink necrotic muscle tissue was seen. This was friable and could be dislodged in clumps with the fingers. The periosteum was greatly thickened, and there was increased vascularity between the periosteum and the femur shaft. In fact, the cortical portion of the femur shaft was so porous that it resembled cancellous bone. After seeing the gross tissue and examining a frozen section, the pathologist stated that this lesion was consistent with a sarcoma and advised radical treatment. This would have been either disarticulation of the hip or a very high thigh amputation, since the whole anterior section of the quadriceps and middle third of the femur shaft were involved. We decided to wait for more detailed study of the pathological sections before proceeding with destructive surgery.

After thorough study of the paraffin sections, the pathologist diagnosed this lesion as the pseudosarcoma stage of myositis ossificans. This was corroborated by others specializing in bone pathology. When the next roentgenogram was made, on April 6, 1951, the diagnosis was very apparent roentgenologically (fig. 2*B*). The typical cloud of well-demarcated ossific material was seen in the muscle mass of the thigh surrounding the femur shaft, the characteristic x-ray appearance of myositis ossificans. Several months later, the roentgenogram showed a definite mass of bone in the quadriceps muscle (fig. 3).

Fig. 3.—Roentgenogram taken in September, 1951, showing definite mass of bone in the quadriceps muscle.

COMMENT

There are several features that prompted reporting this case. The history given by the patient and his family excluded definite knowledge of injury, the one outstanding etiological agent in myositis ossificans circumscripta. Without a history of definite trauma, a rapidly growing bone tumor seemed to be the most possible diagnosis. The age of the patient also was misleading.[3] In the literature reviewed by us, no case of myositis ossificans of the quadriceps was recorded in so young a person. Most cases of myositis ossificans of the quadriceps occurred in the second and third decades.

The early roentgenograms did not help in the diagnosis, for they merely showed a single line of periosteal reaction (fig. 2*A*). This observation can be very misleading. It has been our experience that such a picture indicates only a periosteal irritation, and almost any neoplastic, traumatic, or inflammatory process in the incipient stage can show these periosteal changes.

The course of the fever was another confusing symptom. Fevers of varying degree are expected with bone tumors, especially with Ewing's sarcoma or osteomyelitis; however, it is not generally known that a febrile course can be seen in myositis ossificans. This sign is explained by the fact that necrotic muscle tissue typical of this lesion is being absorbed and causes a foreign protein thermal reaction. Howard,[4] in his report of five cases of myositis ossificans circumscripta in young men in preflight training, noted that each patient ran a fever. We should like to reemphasize that the persistent fever caused us to consider osteomyelitis or Ewing's sarcoma in the differential diagnosis.

Surgical biopsy was the most important diagnostic procedure.[5] We dismiss needle biopsy as useless and misleading. Frozen sections also may be deceiving and hazardous, because osseous tissue cannot be made into good microscopic sections by this technique. Accurate diagnosis requires material that can be examined morphologically and cytologically in order to differentiate neoplastic from inflammatory tissue.

A number of years ago, this patient's leg might have been amputated at once. It was felt then that biopsy of a neoplastic lesion was hazardous because the procedure stirred up the process and spread the tumor. This concept, fortunately, is no longer held by most workers.

Bone lesions are so often equivocal that, when radical treatment is contemplated, a number of opinions must be obtained. In reviewing cases of the Bone Tumor Registry at the American College of Surgeons, Rossiter[6] found three instances among about 700 cases in which apparently clinically proved osteogenic sarcoma was proved to be myositis ossificans on pathological examination.[7] Two of the three patients had amputation. The cases were considered to be five year cures by the submitting physicians. In our judgment, waiting for an accurate diagnosis will not mean the difference between cure and death in true osteogenic sarcoma. The patient should always be given the benefit of all diagnostic procedures before being given radical treatment.

SUMMARY

In a case of myositis ossificans the usual causal factor of definite trauma was lacking. The patient was much younger than is usual in cases of myositis ossificans circumscripta involving the quadriceps muscle. A febrile course is probably a usual observation in the acute phase of myositis ossificans. Surgical biopsy is the most important diagnostic procedure, but it must be interpreted by experts in the field. Amputation to treat a bone tumor should never be done until permanent, good tissue sec-

3. Jones, R., and Morgan, D.: On Osseous Formations in and About Muscles Due to Injury, Arch. Roentg. Ray **10**: 10, 1905-1906. Lewis, D.: Myositis Ossificans, J. A. M. A. **80**: 1281 (May 5) 1923. Magliulo, A.: Contributo allo studio delle ossificazioni muscolari post-traumatiche, sperimentale, Arch. di biol. **34**: 301, 1930. Shipley, A. M.: Ossifying Hematoma and Allied Conditions, Arch. Surg. **41**: 516 (Aug.) 1940.

4. Howard, C.: Traumatic Ossifying Myositis, U. S. Nav. M. Bull. **46**: 724, 1946.

5. Coley, W. B.: Myositis Ossificans Traumatica: A Report of Three Cases Illustrating the Difficulties of Diagnosis from Sarcoma, Ann. Surg. **57**: 305, 1913. Geschickter, C. F., and Maseritz, I. H.: Myositis Ossificans, J. Bone & Joint Surg. **20**: 661, 1938.

6. Rossiter, L. J.: Personal communication to the authors.

7. Chambers, T. R.: Myositis Ossificans: Study of Another Unusual Case, Arch. Surg. **16**: 755 (March) 1928. Pack, G. T., and Braund, R. R.: Development of Sarcoma in Myositis Ossificans, J. A. M. A. **119**: 776 (July 4) 1942.

tions have been examined by competent pathologists. We believe that some of the five year cures reported in osteogenic sarcoma cases may be merely five year survivals of the misdiagnosed pseudosarcoma stage of myositis ossificans.

55 E. Washington St. (2) (Dr. Weinstein).

INVERTED APPENDICAL STUMP SIMULATING CANCER OF THE CECUM

Arkell M. Vaughn, M.D.
and
Jerrold Widran, M.D., Chicago

Roentgenographic diagnosis of abnormality in the cecum can often be misleading. It is not always possible to differentiate neoplastic changes from benign conditions that distort the cecum, even with air contrast studies. The commonest conditions causing difficulty in roentgenographic differential diagnosis in the cecal region are fecal scyballa, distortion secondary to an appendical abscess, regional ileitis, tuberculosis, cecal diverticulitis,[1] amebic involvement of the cecum, ulcerative colitis, adhesions following an appendectomy, and an inverted appendical stump. The case we are reporting is that of patient who had had an appendectomy. An irregular filling defect of the cecum, suggestive of a neoplasm on examination, was found at surgery to be an inverted appendical stump.

Gottlieb and associates [2] emphasized the ever-present danger of misinterpreting feces for an intraluminal defect. Adequate preparation plus air contrast enemas are important. An appendical abscess will give an extraluminal smooth mass but may give enough distortion to make its differentiation from a neoplasm difficult. Regional ileitis, tuberculosis, and adhesions following an appendectomy will, in most cases, involve the ileum as well as distort the cecum. In amebic involvement of the cecum, there is usually evidence of amebic colitis elsewhere.

The presence of an intraluminal defect of the cecum caused by an inverted appendical stump was first described by Artner [3] in 1929. With controlled studies, he demonstrated that more than one-third of inverted appendical stumps are persistent and can be visualized in 8% of cases as isolated ring-like structures. Occasionally it is not possible to differentiate a cecal filling defect caused by an inverted appendical stump from a neoplasm and may exist without producing a palpable mass, change in bowel habits, gross blood in the stool, or loss of weight. An unexplained anemia may be the only clinical sign.

From the departments of surgery, Mercy and Cook County hospitals, and Stritch School of Medicine, Loyola University.

The roentgenographic pictures were made by Dr. Fay Squires, Presbyterian Hospital.

1. Vaughn, A. M., and Narsete, E. M.: Diverticulitis of the Cecum, A. M. A. Arch. Surg. **65**: 763-769 (Nov.) 1952.

2. Gottlieb, C.; Baranbaum, S. L., and Dorfman, M.: Roentgen Features of Non-Malignant Periappendiceal and Ileocaecal Lesions, Am. J. Digest. Dis. **18**: 79-86, 1951.

3. Artner, H.: Wiener Röntgengesellschaft, Fortschritte a. d. Geb. d. Röntgenstrahlen **39**: 148-152, 1929; Der Status Post-Appendectomiam im Röntgenbilde, ibid. **39**: 609-615, 1929.

REPORT OF A CASE

A white woman aged 39 was admitted to Mercy Hospital in October, 1952. In March, 1952, a supracervical hysterectomy had been performed for uterine fibroids. At that time, an appendectomy was done. Since then she had been in good health until two weeks prior to admission, when, because of dizziness, she sought medical aid. On examination by an internist, a small mass was palpated in the right lower quadrant of the abdomen and an associated mild anemia was found. On advice of the internist, a barium enema was done and revealed a filling defect in the cecum. She was subsequently referred to one of us (A. M. V.) for surgery.

Physical examination on admission revealed an apparently healthy woman. The blood pressure was 130/80 mm. Hg, and the heart and lungs were normal. Abdominal examination revealed a lower right rectus scar, no tenderness, and no mass in the right lower quadrant. There were no gastrointestinal symptoms, abdominal pain, or weight loss. Vaginal examination revealed that the cervix was small, scarred, mobile, and not tender. On bimanual examination, no lower abdominal masses were palpated. Laboratory findings showed red blood cells, 3,660,000; white blood cells, 8,100 per cubic millimeter; and hemoglobin 12 gm. per 100 cc. The feces did not contain occult blood. The urine was normal. Barium enema was repeated with air contrast studies, and the cecal filling defect seen two weeks previously was unchanged. A neoplasm could not be ruled out so the patient was prepared for a hemicolectomy on the right side. Succinylsulfathiazole (Sulfasuxidine) was administered for seven days, and neomycin was given for the two days preceding surgery.

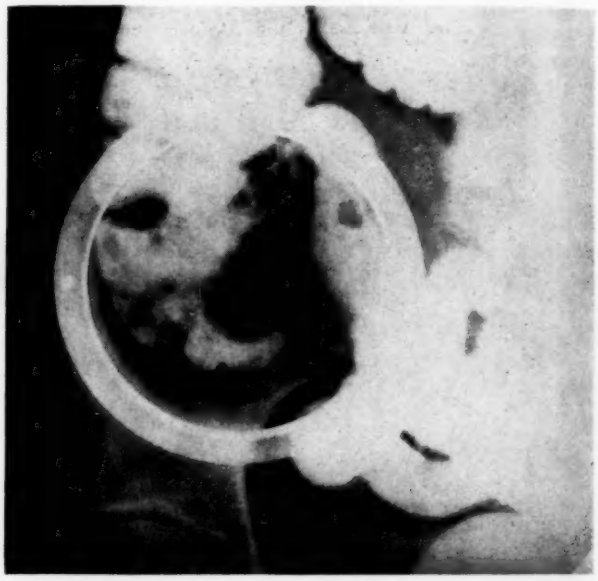


Fig. 1.—Filling defect in the cecum that represents either an appendical stump or a neoplasm.

At surgery, a right paramedian incision removed the old scar. The cecum was dissected free from the surrounding adhesions, brought into the wound, and walled off from the abdominal cavity. External palpation disclosed a small movable mass in the base of the cecum. There were no palpable lymph nodes in the mesentery. The cecum was incised longitudinally through the anterior taenia and the cecal mucosa inspected. The inverted appendical stump was found, but there was no abnormal growth on it or the surrounding mucosa. The stump was excised, and the cecum was closed in two layers.

The pathologist's report stated, "Specimen consists of a mass of tissue measuring 1.5 by 1 by 1 cm. The surface is red and uneven and at one end a small dimple is noted. The other end has been recently sectioned. Section reveals greyish-tan pliable tissue. Microscopic Diagnosis: Inverted stump of appendix. Focal fibrosis and mild focal chronic inflammation."

The postoperative course was uneventful, and the patient was discharged 10 days after surgery. She has been in good health and asymptomatic since. A repeat barium enema, made six months later, showed an absence of the previously irregular ragged defect in the cecum and a smooth filling defect at the site of surgery.

COMMENT

An inverted appendical stump should be considered in the differential diagnosis of a roentgenographic cecal filling defect in every patient who has had an appendectomy. Barium enemas with air contrast studies should be repeated at frequent intervals, and any change in the size of the lesion should be an indication for exploration. A palpable mass in the right lower quadrant, with an associated unexplained anemia, should be an indication for surgery without a prolonged period of observation. Stabins and Dixon [4] state that any unexplained anemia, with few or no symptoms, should make carcinoma of the cecum suspect. As stated previously, a cecal neoplasm may exist without producing a palpable mass, change in bowel habits, gross blood in the stool, or loss of weight. As long as no neoplasm was found on frozen section in our case, it was thought that cutting the protruding tissue flush with the bowel was sufficient, rather than doing a local excision of the cecum.

Fig. 2.—Smooth rounded filling defect in the cecum is a result of surgery.

SUMMARY

Frequent difficulty in making a positive diagnosis of carcinoma of the cecum in the presence of roentgenographic evidence of a cecal filling defect is discussed. Repeated barium enemas with air contrast studies are emphasized as aids in the differential diagnosis of cecal disease. A case in which there was an irregular filling defect of the cecum that was proved at surgery to be an inverted appendical stump is reported.

30 N. Michigan Ave. (2) (Dr. Vaughn).

4. Stabins, S. J., and Dixon, J. A.: Silent Carcinoma of Cecum, New York J. Med. 51: 2140-2146, 1951.

FATAL HEMORRHAGE FOLLOWING NEEDLE BIOPSY IN UREMIA

REPORT OF A CASE

Samuel Zelman, M.D., Topeka, Kan.

I have encountered only one serious hemorrhage following needle biopsy in over 600 such biopsies of the liver and other organs or tumor masses. In this case, the patient's prognosis was hopeless in any event. However, the experience suggests the advisability of listing uremia among the contraindications to needle biopsy or at least suggests that the increased hazard of hemorrhage in uremia be carefully considered.

REPORT OF A CASE

A 54-year-old white married male rubber factory worker was admitted to Winter Veterans Administration Hospital on Dec. 19, 1952. He stated he had been feeling weak and tired for the past year. Two weeks before admission, he had had an upper respiratory infection together with a periodontal abscess. The diseased tooth was extracted after four days of penicillin therapy. Since then, he had become steadily worse, with anorexia, morning nausea and retching with no vomiting, slight fever, a constant sense of fatigue, headache, shortness of breath on exertion, and intermittent sharp epigastric pain. During the week past, he had been constantly drowsy and unable to work. Diminished excretion of urine, which was dark brown and smoky in appearance, had been present for five days. His fluid intake had been no more than three or four glasses of water per day. For the past four days he had noted morning swelling of the face and hands, and swelling of the feet and ankles during the day. The day before admission, he thought his skin had a yellow tinge. He had lost 10 lb. (4.5 kg.) during the preceding two weeks. There had been no change in the color of his stools, no nocturia, and no nocturnal dyspnea. His personal habits, his history, and his family history were noncontributory. No history of exposure to toxic substances could be obtained, other than his employment handling rubber tires and the occasional use of mercurous chloride (calomel).

The patient was a well-nourished and well-developed white man who appeared moderately ill. He was drowsy but oriented and cooperative. He was 70 in. (1.77 m.) tall and weighed 130 lb. (59 kg.). His usual weight had been 145 lb. (65.8 kg.) prior to the present illness. His temperature was 99.8 F, his pulse rate 60 per minute with occasional extrasystoles, and his blood pressure 136 mm. Hg systolic and 86 mm. Hg diastolic. The skin and scleras were mildly icteric. The pupils reacted normally, extraocular movements were intact, and the ocular fundi were not abnormal. The remaining teeth appeared in fair condition. The chest was symmetrical, with bilaterally equal and adequate expansion. The lung fields were clear to percussion, but fine moist rales were audible at both lung bases posteriorly. The heart was not abnormal. The abdomen was soft. A smooth, tender liver edge was palpable 5 cm. below the right costal margin. Bilateral tenderness in the loin could be elicited by kidney punch. There was slight edema of the face and lower legs. Tendon reflexes were hyperactive. There was no lymphadenopathy.

The leukocyte count on admission was 7,500 per cubic millimeter, with a normal differential count. Erythrocytes numbered 4,100,000 per cubic millimeter. The hemoglobin level was 11.7 gm. per 100 cc. (Haden-Hausser method), the hematocrit was 36%, and the corrected sedimentation rate was 32 mm. per hour (Wintrobe method). Three blood cultures produced no growth. Serum agglutination tests for typhoid H and O antigens, paratyphoid A and B, Brucella abortus, Pasteurella tularensis, Proteus OX19, and heterophil antibody were not significant. Serologic tests for syphilis were negative. Bleeding, clotting, clot retraction, and prothrombin times were within normal limits.

From the Medical Service, Winter Veterans Administration Hospital.
This case is reported with the permission of Dr. Joseph E. Smelser of the Medical Service and Dr. Alfred Plaut, pathologist.

The urine was acid, ranged in specific gravity from 1.010 to 1.019, was negative for sugar and bromide, and showed albumin (4+), and macroscopically visible blood with numerous white and red cells visible microscopically, as well as variable numbers of hyaline, granular, and white and red blood cell casts. Tests for heavy metals in the urine were negative. Urine urobilinogen was reported present in high concentration on a single determination. The blood urea nitrogen ranged between 63 and 66 mg. per 100 cc. during the first week of hospitalization. The serum chlorides were 90 mEq. per liter, and the carbon dioxide combining power of the plasma 46 vol. %. The serum bilirubin was 0.2 mg. per 100 cc., the serum alkaline phosphatase was 6.6 Bodansky units per 100 cc., and the inorganic phosphorus content was 6.1 mg. per 100 cc. The cephalin-cholesterol flocculation test was negative, but the sulfobromophthalein sodium (Bromsulphalein) retention at 45 minutes was 17% (5 mg. per kilogram of body weight injected).

Roentgenograms of the chest were taken Dec. 22, 24, and 29. In comparison with a roentgenogram taken in 1948, these showed consistently higher diaphragmatic levels, with widening of the heart shadow presumed to be positional. In addition, infiltrations of somewhat homogeneous appearance were seen in both lower lung fields, varying in location and extent among the films. These presumably represented areas of uremic congestion and edema, although they were interpreted at the time as atypical pneumonias and effusions. An abdominal roentgenogram proved noncontributory. Electrocardiograms taken Dec. 20 and 30 showed slurring and low voltage of the ventricular complexes. The second electrocardiogram showed, in addition, lower and broader P waves, with increase in the P-R interval from 0.16 to 0.24 second, and widening of the QRS from 0.09 to 0.11 second, suggesting the development of myocardial conduction difficulty.

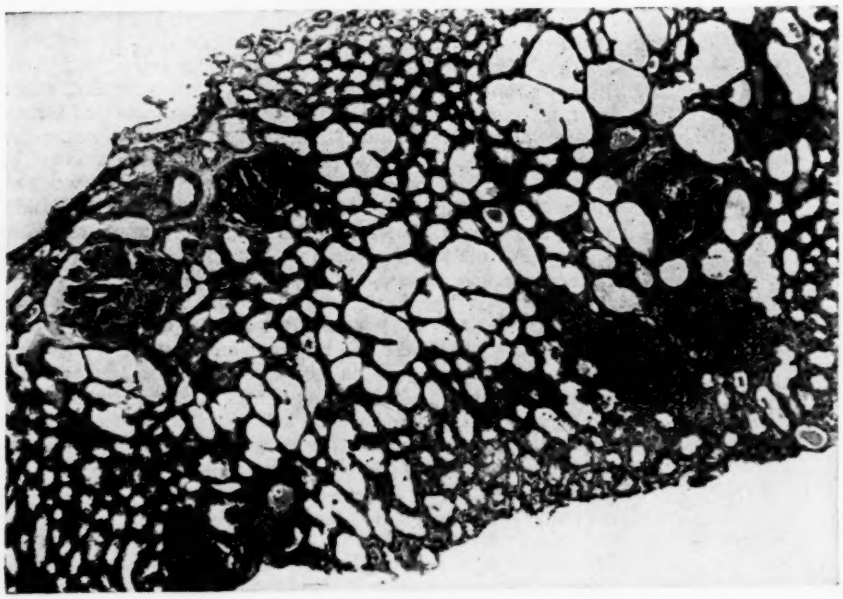


Fig. 1.—Acute glomerulonephritis, section of needle biopsy of kidney, hematoxylin and eosin stain (× 59).

The patient was confused, restless, and drowsy throughout his hospital course. Nausea, vomiting, and diarrhea were intermittent. Abdominal and low back pain and chilliness were noted by the patient. Remittent fever, with temperature as high as 100 F, was present during the first four days of hospitalization, after which the patient was afebrile. Oliguria was present throughout the hospital course, varying from a 24 hour maximum of 370 cc. to a minimum of 60 cc., in general gradually decreasing in quantity until death. Blood pressure remained at normal levels. Muscular twitchings appeared Dec. 28.

Clinical observations pointed to acute hepatitis and a lower nephron nephrosis. The patient was digitalized, and chlortetracycline (Aureomycin) and penicillin were administered. Fifteen per cent dextrose infusions were repeatedly administered intravenously, with no effect on the oliguria. On Dec. 29, a leukocyte count of 11,850 per cubic millimeter was recorded, with 82% granulocytes. The blood urea nitrogen was now 136 mg. per 100 cc., the creatinine was 12.9 mg. per 100 cc., the serum chloride was 72 mEq. per liter, and the carbon dioxide combining power was 30 vol. %. The serum albumin was 3.3 gm. and the globulin was 2.2 gm. per 100 cc., the calcium was 10 mg. per 100 cc. and phosphorus was 12.4 mg. per 100 cc. The following day, serum sodium was 121.5 mEq. per liter and potassium was 8.2 mEq. per liter. An attempt was made to counter hyponatremia by intravenous infusion of 5% saline solution, of which 200 cc. was administered Dec. 29 and 125 cc. was given Dec. 30.

The patient appeared to be rapidly approaching death, and the impatiently awaited diuresis heralding recovery from what was assumed to be lower nephron nephrosis did not materialize. As a last measure, to help in arriving at a more definitive diagnosis, needle biopsies of the right kidney and of the liver were done at 2:30 p. m. on Dec. 30. Technique on this occasion was flawless; the patient cooperated well despite his stupor, and excellent specimens were obtained bloodlessly with a single puncture of each organ. A light scratching on the surface of the liver was deliberately produced before penetration of the liver was attempted. This maneuver was designed to gage by the sense of touch the extent of possible laceration if puncture should be carried out during respiration because of the patient's stuporous mental state. The scratches thus defined the respiratory excursion of the liver at this point. The excursion was deemed excessive, and the needle was withdrawn until the patient proved he could cooperate by holding his breath if continuously exhorted to do so throughout the procedure. After that, needle puncture was uneventful. An intercostal approach was used. After the biopsy the patient gradually lapsed into coma, and he died at midnight. Hemorrhage was not suspected from the clinical observations.

The needle biopsies revealed only secondary minor hepatic changes but revealed the presence of severe acute and subacute glomerulonephritis (fig. 1), making evident the hopelessness of the prognosis in the light of the patient's clinical course. At autopsy, the abdominal cavity was found to be filled with 2 liters of fluid and clotted blood. Bilateral pleural effusions were not bloody. Right perirenal hemorrhagic infiltration was present, possibly amounting to half a liter of blood. The puncture wounds in the convexity of the right lobe of the liver and in the posterior surface of the right kidney were no larger than needle size and showed no surrounding hemorrhagic infiltration of the capsules or parenchyma (fig. 2). On section, the needle tracks were evident as narrow streaks without surrounding hemorrhage. Neither puncture entered any macroscopic vessel or duct, nor did the renal puncture reach the pelvis or calix. It was evident that bleeding in both instances was a result of defection of hemostasis rather than of trauma.

Other observations of interest at autopsy included rheumatic myocardial lesions of varying ages, subendocardial and gastric mucosal hemorrhages, generalized and cerebral edema of moderate degree, persistence of the thymus, and two necrotic foci in the anterior pituitary. The pituitary necroses were 2 to 4 and 7 to 10 days old, according to Sheehan's criteria,[1] and therefore unrelated to the terminal hemorrhage. The liver showed a moderate degree of central stasis and some increase in the portal connective tissue.

The kidneys were large, pale, and smooth, with many sharply circumscribed small surface hemorrhages. They were soft and soggy in texture. The cortices were slightly wider than normal, with fairly distinct striations and with normal outlines of the slightly darker pyramids. The glomeruli were barely visible, and

1. Sheehan, H. L.: Post-partum Necrosis of the Anterior Pituitary, J. Path. & Bact. 45: 189-214, 1937.

the papillae were not abnormal. The mucosae of the pelves and calices were irregularly but widely hemorrhagic. Histologically, most of the glomeruli were bloodless; some were filled with leukocytes. Crescents were few. In some glomeruli the vascular loops showed little change, but the capsule was notably thickened next to the vascular pole. There was much interstitial inflammation, consisting mostly of mononuclear cells. Many tubules contained blood and fibrin. Other than the presence of the needle track, there were no gross or microscopic differences between the two kidneys.

COMMENT

For two liters of blood to have spilled into the peritoneal cavity, one must envision a stream of blood amounting to 4 cc. per minute continuously during the nine and one-half hours of the patient's survival. The minor trauma of a puncture wound no more than 3 mm. in diameter cannot by itself account for such hemorrhage. A defect of hemostasis must have been present. The coexistence of hemorrhage from the similarly small needle wound of the kidney, in which less blood was lost presumably because of the lesser distensibility of the perirenal tissues, would appear to confirm the existence of such a defect. The needles entered no large blood vessels. The bleeding time, coagulation time, clot retraction time, and prothrombin time of this patient were determined to be within normal limits. Persistent oozing had not occurred following venipunctures, nor did any occur from the skin incisions of the two needle biopsies. Capillary fragility was not determined. Although hemorrhages were later found in the subendocardial area, stomach mucosa, and renal pelves, there had been no visible evidence of hemorrhages as petechiae or ecchymoses or of hemorrhagic discharges other than the hematuria connected with the nephritis and a brief and minor epistaxis that occurred Dec. 23.

Uremia has not previously been listed as a contraindication to the performance of needle biopsies.[1a] Indeed, it is rarely listed in classifications of the causes of hemorrhage.[2] Nevertheless, hemorrhage as a complication of nephritis and uremia has been familiar to clinicians since the days of Richard Bright (1827). Epistaxis and retinal and cerebral hemorrhages are often observed. In a review of the hemorrhagic manifestations sometimes seen in nephritis, Riesman [3] also listed metrorrhagia, hemoptysis, hematemesis, and hemorrhage into the ear. Mucosal hemorrhages in the gastrointestinal tract in cases of uremia were reported at autopsy in 52.9% of 136 cases by Jaffe and Laing [4] and in 21.1% by Mason.[5] These mucosal hemorrhages are the commonest digestive tract lesions seen in uremia. Hemorrhagic pleurisy and pericarditis may occur. Mediastinal hemorrhage has been reported.[5a] Jaffe and Laing [4] mention also hemorrhages in the epicardium, lung, mucosa of the renal pelvis, urinary bladder, uterus, and vagina. Six of their patients showed so many petechiae of the skin that the diagnosis on admission suggested a blood dyscrasia. Generalized purpura involving the skin and mucous membranes is seen occasionally as a terminal sign in uremia. Riesman [3] reported two such cases. Davidson [6] observed three instances of purpura in chronic interstitial nephritis associated with marked elevations of blood urea levels, reported as 245, 360, and 428 mg. per 100 cc.

Streicher [7] produced a uremia-like condition in dogs by the slow intravenous injection of large amounts of urea and observed a congested and hemorrhagic intestinal mucosa. Bollman and Mann [8] produced hemorrhagic lesions in the stomach that were associated with marked elevations of blood urea levels by transplantation of the ureters into the intestine of dogs.

The mechanism of the hemorrhagic state occurring in uremia has never been clearly elucidated. Vascular degeneration and hypertension are not necessary factors. In a consideration of the possible role of arteriolosclerotic changes in the intestinal tract as a basis for the hemorrhages in uremia, Jaffe and Laing [4] found that vascular necrosis was always in close proximity to the necrosis of the mucosa and was never observed during the congestive or hemorrhagic stage of the lesions. The vascular necrosis was distinctly inflammatory and thus undoubtedly secondary, as is seen commonly in bacillary dysentery.

Schoenemann [9] reported the postmortem observation in persons with nephritis of numerous superficial hemorrhages into the nasal mucosa. The capillaries near the

Fig. 2.—Photograph of a portion of the surface of the right lobe of the liver, showing the site of needle puncture and superficial scratching of the liver capsule.

epithelial surface were increased in number and in many cases had broken through the basement membrane, the capillary loops having forced their way through the basal epithelial cells. The capillary walls appeared normal, and the deeper vessels showed no degenerative changes. These

1a. Terry, R.: Risks of Needle Biopsy of the Liver, Brit. M. J. **1**: 1102-1105, 1952.

2. Wintrobe, M. M.: Clinical Hematology, ed. 3, Philadelphia, Lea & Febiger, 1951, p. 255.

3. Riesman, D.: Hemorrhages in the Course of Bright's Disease with Special Reference to the Occurrence of a Hemorrhagic Diathesis of Nephritic Origin, Am. J. M. Sc. **134**: 709-716, 1907.

4. Jaffe, R. H., and Laing, D. R.: Changes of the Digestive Tract in Uremia, Arch. Int. Med. **53**: 851-864, 1934.

5. Mason, E. E.: Gastrointestinal Lesions Occurring in Uremia, Ann. Int. Med. **37**: 96-105, 1952.

5a. Brown, N.; Tomsykoski, A. J., and Stevens, R. C.: Mediastinal Hemorrhage Secondary to Uremia, Am. J. Med. **15**: 588-590, 1953.

6. Davidson, L. S. P.: Classification and Treatment of the Purpuras, Proc. Roy. Soc. Med. **30**: 715-723, 1937.

7. Streicher, M. H.: Experimental Uremia: Uremic Enteritis, Arch. Int. Med. **42**: 835-845, 1928.

8. Bollman, J. L., and Mann, F. C.: Nitrogenous Constituents of Blood Following Experimental Transplantation of Ureters into Different Levels of Intestine, Proc. Staff Meet., Mayo Clin. **2**: 134-136, 1927.

9. Schoenemann, A.: Arch. Laryng. u. Rhin. **12**: 437, 1901-1902; quoted by Riesman.[3]

changes were not observed in diseases other than nephritis. Riesman [3] suggested that the changes were due to a circulating substance that weakened the capillary walls and surrounding structures.

Jaffe and Laing,[4] in their studies of the digestive tract in uremia, could detect no alterations of the walls of the capillaries or small veins, except for extreme dilatation of these vessels in contrast to narrow arterioles and small arteries. They suggested that the hemorrhages might be the result of disturbances in the innervation of the terminal vascular bed. They said that the increased permeability that is associated with the extreme widening of the small blood vessels leads first to edema and later to hemorrhages. Ulceration was attributed to bacterial invasion of the devitalized hemorrhagic areas.

No consistent coagulation defect has been demonstrated in the reported instances of uremic hemorrhage. Boyd [10] states that there is a curious tendency toward hemorrhages in uremia for no apparent reason and that there is no satisfactory explanation beyond the vague suggestion of some toxic action on the vessel walls. Moore [11] states that the purpura of uremia has never been adequately explained and suggests that, since it is known that there is material damage to the liver in uremia, hypoprothrombinemia may be a cause. Atchley [12] reports that an elevated prothrombin time has been observed in a number of uremic patients at the Presbyterian Hospital in New York who showed hemorrhagic tendencies. On the other hand, Fishberg [13] and Allen [14] state that the coagulation time of the blood is usually increased in uremia. Davidson [6] found no reduction in the number of platelets in two out of three cases in which examinations were made. A decreased capillary resistance in some cases of chronic renal disease has been reported.[15]

It is apparent that a study of hemostasis in uremia is needed. A consideration of the studies of Streicher,[7] Schoenemann,[9] and Jaffe and Laing [4] leads to the conjecture that an increase in capillary fragility may prove to be a clinically demonstrable defect in uremic patients. Capillary fragility was not determined in the case reported above. Had it been determined and found appreciably increased, the needle biopsies would not have been performed. Since this experience, it has been my invariable practice to add the capillary fragility determination to the bleeding, clotting, clot retraction, and prothrombin times done prior to needle biopsy. A few instances have been found, in the absence of uremia, in which increased capillary fragility seemed to contraindicate biopsy, although other tests of hemostasis were normal.

SUMMARY

In a case of uremia, hemorrhages followed nonlacerating needle biopsies of liver and kidney. Needle biopsy may be contraindicated in some instances of uremia.

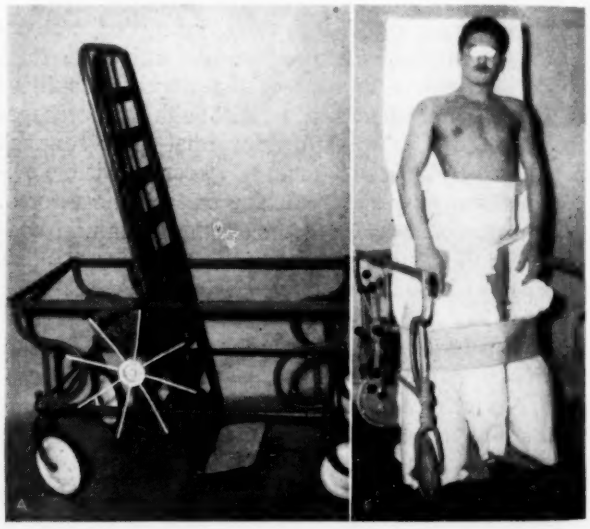
10. Boyd, W.: The Pathology of Internal Diseases, ed. 5, Philadelphia, Lea & Febiger, 1951, p. 421.
11. Moore, R. A.: A Textbook of Pathology, ed. 2, Philadelphia, W. B Saunders Company, 1951, p. 756.
12. Atchley, D. W.: Uremia, in Cecil, R. S., and Loeb, R F.: A Textbook of Medicine, ed. 8, Philadelphia, W. B. Saunders Company, 1951, p. 956.
13. Fishberg, A. M.: Hypertension and Nephritis, ed. 4, Philadelphia, Lea & Febiger, 1939, p. 182.
14. Allen, A. C.: The Kidney: Medical and Surgical Diseases, New York, Grune & Stratton, Inc., 1951, p. 68.
15. Schultzer, P.: Studies on Capillary Resistance, Acta med. scandinav **81**: 113-132, 1934.

USE OF A TILT-TABLE IN EARLY REHABILITATION OF PARAPLEGIC PATIENTS

Samuel Climo, M.D., New Haven, Conn.

In discussing the prophylaxis of decubitus ulcers in paraplegic patients, it must be admitted that the ulcers develop in patients in every hospital regardless of size and over-all efficiency. The earlier the patient leaves his bed and avoids or reduces the complications of prolonged immobilization, namely, bedsores, decalcification of bones, and genitourinary infections, the better are his chances for rehabilitation, both occupational and psychological. The opportunities for rehabilitation are made available early by the use of a modified, four-wheeled stretcher that is capable of securing and maintaining the paraplegic patient in the erect posture (see figure) as soon as the medical status permits.

A patient can remain strapped in the erect position for many hours, since the plantar integuments, composed of highly specialized tissue, are capable of withstanding prolonged pressure from body weight. Thus, a patient can engage in skillful occupation. Since the table rotates



A, modified four-wheeled stretcher with the top tilted vertically and. *B*, patient strapped to the top and tilted.

from a horizontal position to a vertical one, any intermediate incline can be used for resting, even sleep. The displacement of floor space by the converted table does not exceed appreciably that of the ordinary wheelchair.

The tilt-table is not available yet commercially. The ordinary four-wheeled stretcher can be converted in any institutional workshop. The horizontal top of the stretcher can be removed and the width of the frame increased to permit the top to rotate on a horizontal axis. Placing the axis off center permits the top to rotate through an angle approximating 90 degrees to the floor. When the tilt-table is in the vertical position, the broad footrest terminates just above the level of the floor. Quarter gears are welded on the underside of the top to rotate on a pivoting axis. A simple gear-locking mechanism is employed to maintain the incline at any level.

291 Whitney Ave.

Plastic Surgeon, Hospital of St. Raphael.

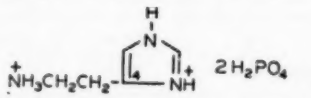
COUNCIL ON PHARMACY AND CHEMISTRY

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. STORMONT, M.D., *Secretary.*

Histamine Phosphate-U.S.P.—The structural formula of histamine phosphate may be represented as follows:



Actions and Uses.—Histamine exists in various organs and tissues of the body, probably in an inert form. It produces local vasodilatation when released from the cell under appropriate stimuli, such as trauma, shock, and possibly allergic reactions. When injected into an animal, histamine stimulates gastric secretion and produces flushing, nausea, bronchospasm, fall in blood pressure, arrhythmia, and gastrointestinal contraction. It acts directly on the receptive substance in smooth muscle.

Histamine, although absorbed orally, produces highly variable effects when administered by this route. Salts of histamine are usually administered subcutaneously, intravenously or intramuscularly.

Histamine phosphate is employed as a test for gastric secretory activity. It also produces temporary benefit in some patients with Ménière's syndrome, including those showing sudden deafness. It has been used in the treatment of multiple sclerosis; although the effects are equivocal, they deserve further study. Some patients apparently experience temporary amelioration of the disease after histamine therapy.

Although histamine has been recommended for treatment of migraine and certain cephalgias, the evidence of value is not convincing. Because of the known hazards of histamine therapy, the drug should not be used indiscriminately in these conditions.

Histamine is a potent drug, and overdosage or administration to susceptible persons may give rise to serious reactions. Vasomotor collapse, shock, and even death may occur quickly if the drug is administered too rapidly or in too great a quantity. Thus, when calculating dosages, it should be remembered that the salt contains only about 36% of the active base. Epinephrine hydrochloride is the antidote of choice in histamine overdosage and should be given intramuscularly or, in severe poisoning, intravenously. A solution of epinephrine hydrochloride 1:1,000 should always be readily available at the time histamine is administered.

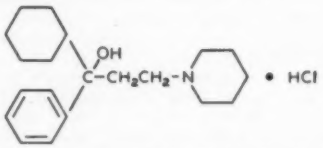
Dosage.—For the treatment of Ménière's syndrome and multiple sclerosis, a slow intravenous injection of histamine phosphate, 1.1 mg. per 100 cc., in isotonic sodium chloride solution may be administered. The initial rate of administration should not exceed 20 to 30 drops per minute and should never exceed 50 to 60 drops per minute. A maximum of 250 cc. of such a solution may be administered in not less than 90 minutes. The therapy may be repeated daily until improvement is noted or until it is determined that the patient will not respond.

Any reaction should be treated immediately with the intramuscular or intravenous injection of 1:1,000 epinephrine hydrochloride.

Don Baxter, Inc., Glendale, Calif.

Solution Histamine Phosphate: 250 cc. Vacoliter bottles. A solution in isotonic sodium chloride containing 1.1 mg. of histamine phosphate in each 100 cubic centimeters.

Trihexyphenidyl Hydrochloride.—Artane Hydrochloride (Lederle).—$C_{20}H_{31}NO.HCl$.—M.W. 337.92.—3-(1-Piperidyl)-1-phenyl-1-cyclohexyl-1-propanol hydrochloride.—The structural formula of trihexyphenidyl hydrochloride may be represented as follows:



Actions and Uses.—Trihexyphenidyl hydrochloride, a synthetic piperidyl compound, exhibits an atropine-like action and exerts an antispasmodic effect on smooth muscle and an inhibitory action on the parasympathetic nervous system. Thus, spasm of smooth muscle is relieved by direct action and by indirect parasympathetic release. The drug also relieves spasticity of voluntary muscle, partly because of its parasympatholytic action and partly because of action on the cerebral motor centers. Unlike atropine, the action of trihexyphenidyl is strongest in producing desirable, relaxant effects and weakest in producing undesirable side-effects. Compared with atropine, trihexyphenidyl produces about one-half as intense antispasmodic action, one-third as great mydriatic action, one-eighth as much antisalogogue effect, and one-tenth as much cardiovagal inhibition.

Trihexyphenidyl hydrochloride is useful for the treatment of all forms of parkinsonism, including the postencephalitic, arteriosclerotic, and idiopathic types. It reduces the muscular rigidity and relieves the depression and mental inertia characteristic of this syndrome. The drug is especially effective in reducing the rigidity produced by muscle spasm, thus increasing the ability of the patient to achieve coordination of muscular motions. Tremor is usually reduced, but in some patients who have been severely spastic, it may become more perceptible as spasticity is relieved. Sialorrhea is reduced, but with less accompanying mouth dryness, blurred vision, or mydriasis than with the use of atropine. Trihexyphenidyl is particularly useful in the treatment of arteriosclerotic parkinsonism because, unlike atropine, it usually does not tend to precipitate glaucoma.

Trihexyphenidyl hydrochloride thus far has seldom produced severe reactions with therapeutic dosages. Some patients experience minor side-effects such as dryness of the mouth, blurring of vision, dizziness, mild nausea, or nervousness. Compared with the symptoms of unrelieved parkinsonism, they are usually not troublesome and may be controlled by adjustment of dosage or time of administration. These side-effects tend to decrease with continued use of the drug. The infrequent but more severe reactions of mental confusion, agitation, or nausea with vomiting tend to occur in arteriosclerotic patients or in persons exhibiting other drug idiosyncrasies. Such patients must develop tolerance to the drug gradually, beginning with a smaller initial dose and increasing the dosage more slowly until an effective level is reached. If a severe reaction occurs, administration of the drug should be discontinued temporarily for several days and then resumed at a lower dosage level. Hypertension or cardiac, liver, or kidney disorders do not contraindicate use of the drug, but patients with these conditions should be carefully observed.

Dosage.—Trihexyphenidyl hydrochloride is administered orally. The usual initial dose is 1 mg. for the first day. If the patient is already receiving treatment with other agents, this initial dose should be substituted for a part of the current therapy. As the dosage of trihexyphenidyl is gradually increased, other medication should be decreased until the drug has replaced the former treatment or until an effective balance has been achieved. With prior therapy and in arteriosclerotic or sensitive patients, daily increments of the dose should be small until satisfactory tolerance is attained. If prior medication or unusual reactivity is not involved, the dosage is increased to 2 mg. for the second day, with subsequent increments of 2 mg. daily until a total daily amount of 6 to 10 mg. is reached. Postencephalitic patients may require as much as 12 to 15 mg. daily. At the lower level of daily dosage, the total amount can be divided into three equal parts, taken near meal times; at the higher level, a fourth dose at bedtime is required. Patients are allowed to choose whether to take the medication before or after meals. Postencephalitic patients, who have more excessive

salivation, will prefer administration after meals and may require small doses of atropine sulfate as an adjuvant. Whenever the mouth becomes excessively dry, the drug can be taken before meals unless this causes nausea; if it is necessary to administer the dose after meals, dryness can be allayed by hard candy, gum, or extra intake of fluid.

Tests and Standards.—

Physical Properties: Trihexyphenidyl hydrochloride is a white, odorless solid, m.p. 249.0-249.5° (with slight decomposition). It is freely soluble in methanol, and very slightly soluble in ether and in benzene. The approximate amounts which dissolve at 25° in the following solvents to form 100 ml. of solution are: 6 gm. in alcohol, 5 gm. in chloroform, and 1 gm. in water. The pH of a 1% solution is 5.5-6.0.

Identity Tests: Dissolve about 50 mg. of trihexyphenidyl hydrochloride in 25 ml. of water. To 5 ml. of the solution add dropwise a saturated aqueous solution of picric acid: a yellow precipitate forms. To another 5 ml. portion add 1 ml. of diluted nitric acid and 1 ml. of silver nitrate T.S.: a white precipitate forms which is soluble in excess ammonia T.S. *(presence of chloride).* To another 5 ml. portion of the solution add 1 ml. of diluted hydrochloric acid and 2 ml. of potassium ferrocyanide T.S.: a light green precipitate forms on standing. To another 5 ml. portion add dropwise 0.1 *N* sodium hydroxide: a white precipitate forms.

Transfer to a 50 ml. volumetric flask 25 mg. of trihexyphenidyl hydrochloride, fill to the mark with 0.1 *N* hydrochloric acid, and mix. Measure the absorbancy in a 1 cm. quartz cell, using 0.1 *N* hydrochloric acid as a blank. The solution exhibits ultraviolet absorption maxima at about 252 and 257 mμ, a minimum at about 254 mμ, and a point of inflection at about 262 mμ.

Purity Tests: Dry about 1 gm. of trihexyphenidyl hydrochloride, accurately weighed, at 105° for 4 hours: the loss in weight is not more than 2.0%.

Char about 0.4 gm. of trihexyphenidyl hydrochloride, accurately weighed, over a low flame, cool the residue, add 1 ml. of sulfuric acid, heat cautiously until evolution of sulfur trioxide ceases, ignite, cool, and weigh: the residue does not exceed 0.05%.

Assay: (Trihexyphenidyl Hydrochloride) To prepare a standard, recrystallize the trihexyphenidyl hydrochloride as follows. To 5 ml. of a saturated solution of trihexyphenidyl hydrochloride in methanol add petroleum ether slowly with vigorous stirring until precipitation is complete. Filter through a fritted glass filter, and wash the precipitate with three 5 ml. portions of a 1:1 mixture of methanol and petroleum ether. Repeat the recrystallization and dry in a vacuum over phosphorus pentoxide at room temperature for 24 hours. Dissolve 0.1 gm. of the recrystallized trihexyphenidyl hydrochloride, accurately weighed, in 5 ml. of methanol. Transfer the solution to a 100 ml. volumetric flask, fill to the mark with water, and mix. Transfer to separate 100 ml. beakers 3, 5, 10, and 15 ml. of the solution. Add 1 ml. of methanol and enough water to dilute the solutions in each beaker to 20 ml. Add 6 ml. of freshly prepared 2% ammonium reineckate in methanol. Heat on a steam bath about 30 seconds. Cool to room temperature with intermittent swirling to induce crystallization. If crystallization does not occur on cooling, reheat the solution. Refrigerate the mixture for 2 hours and filter with suction, collecting the precipitates in fritted glass funnels. Wash the precipitates with 5 ml. portions of ice-cold water until the filtrates are colorless. Place test tubes under the stems of the funnels in the suction flasks, and, with the aid of suction, wash the precipitates into the test tubes with 2 ml. portions of acetone until the precipitates are entirely dissolved. Transfer the acetone solutions to 10 ml. volumetric flasks, fill to the marks with acetone, and mix. Spectrophotometrically determine the absorbancies in 1 cm. cells at 526 mμ, using acetone as a blank. Plot the absorbancies against concentrations to give a standard curve. Dissolve about 0.1 gm. of the trihexyphenidyl hydrochloride to be analyzed, accurately weighed, in 5 ml. of methanol. Transfer the solution to a 100 ml. volumetric flask, fill to the mark with water, and mix. Transfer to a 100 ml. beaker 10 ml. of the solution, add 1 ml. of methanol and 9 ml. of water, and proceed as before. Read the concentration of trihexyphenidyl hydrochloride from the standard curve. The amount of anhydrous trihexyphenidyl hydrochloride is not less than 98.0 nor more than 102.0%.

Dosage Forms of Trihexyphenidyl Hydrochloride

ELIXIRS. *Identity Tests:* To 100 ml. of the elixir add with stirring 50 ml. of ammonia T.S. and allow the mixture to stand for 30 min. Using suction, filter the mixture through a fritted glass filter funnel. Wash the precipitate with three 5 ml. portions of water. Place a test tube under the stem of the funnel in the suction flask and, with the aid of suction, wash the precipitate into the test tube with five 3 ml. portions of 0.1 *N* hydrochloric acid. Transfer the solutions to a 50 ml. volumetric flask, fill to the mark with 0.1 *N* hydrochloric acid, and mix. The solution responds to the spectrophotometric identity test for the active ingredient in the monograph for trihexyphenidyl hydrochloride.

Assay: (Trihexyphenidyl Hydrochloride) Transfer to a 125 ml. Erlenmeyer flask a volume of elixir, accurately measured, equivalent to about 10 mg. of trihexyphenidyl hydrochloride, add 10 ml. of diluted hydrochloric acid, and proceed with the spectrophotometric assay as directed in the monograph for trihexyphenidyl hydrochloride starting with, "Add 6 ml. of freshly prepared 2% ammonium reineckate in methanol." The amount of trihexyphenidyl hydrochloride is not less than 95.0 nor more than 105.0% of the labeled amount.

(Alcohol) To 100 ml. of elixir add 100 ml. of water and on the solution run a *U. S. P. alcohol determination.* The amount of alcohol is not less than 90.0 nor more than 110.0% of the labeled amount.

TABLETS. *Identity Tests:* Powder a number of tablets equivalent to about 25 mg. and triturate the powder with 10 ml. of 0.1 *N* hydrochloric acid. Transfer the mixture to a 15 ml. centrifuge tube and centrifuge for 10 min. at about 2,500 rpm. With the aid of suction, filter the supernatant liquid through a fritted glass filter funnel into a test tube placed under the stem of the funnel in the suction flask. Add 10 ml. of 0.1 *N* hydrochloric acid to the centrifuge tube, break up the residue at the bottom of the tube, and stir thoroughly. Centrifuge the mixture again and filter the supernatant liquid. Transfer the filtrate to a 50 ml. volumetric flask, fill to the mark with 0.1 *N* hydrochloric acid, and mix. The solution responds to the spectrophotometric identity test for the active ingredient in the monograph for trihexyphenidyl hydrochloride.

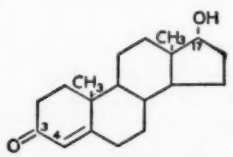
Assay: (Trihexyphenidyl Hydrochloride) Weigh 20 tablets and powder them. Transfer to a 50 ml. centrifuge tube an amount of powder, accurately weighed, equivalent to about 20 mg. of trihexyphenidyl hydrochloride. Add 5 ml. of water and heat in a steam bath for 30 min., stirring the mixture until the powder is thoroughly wetted. Remove the mixture from the bath, add 15 ml. of methanol, and stir the mixture until it is thoroughly wetted. Place the tube in a steam bath, stir the mixture continuously, and allow the methanol to boil vigorously about 2 minutes. Centrifuge the mixture for 5 minutes at about 3,000 rpm. Pour the supernatant liquid into a 250 ml. beaker containing several glass beads. Repeat the extraction with four additional 15 ml. portions of methanol. Evaporate the combined methanol extracts to about 5 ml. and add 20 ml. of water. Transfer the solution to a 50 ml. volumetric flask, fill to the mark with water, and mix. Transfer 20 ml. of this solution to a 100 ml. beaker and proceed with the spectrophotometric assay as directed in the monograph for trihexyphenidyl hydrochloride starting with, "Add 6 ml. of freshly prepared 2% ammonium reineckate in methanol." The amount of trihexyphenidyl hydrochloride is not less than 95.0 nor more than 105.0% of the labeled amount.

Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y.

Elixir Artane Hydrochloride: 473 cc. and 3.78 liter bottles. A flavored elixir containing 0.5 mg. of trihexyphenidyl hydrochloride in each cubic centimeter. Preserved with 0.08% methylparaben and 0.02% propylparaben.

Tablets Artane Hydrochloride: 2 and 5 mg. U. S. trademark 500,574.

Testosterone-U.S.P.—Androlin (Lincoln).—$C_{19}H_{28}O_2$.—M.W. 288.41.—The structural formula of testosterone may be represented as follows:



Actions, Uses, and Dosage.—Testosterone in aqueous suspension apparently has a slightly lesser intensity and slightly greater duration of androgenic action than testosterone propionate. See monograph on testosterone propionate in N. N. R.

Tests and Standards.—

Dosage Forms of Testosterone

SUSPENSIONS. *Identity Tests:* Filter through a fritted glass funnel of fine porosity an amount of suspension equivalent to about 0.15 gm. of testosterone. Wash the residue with two 10 ml. portions of water and dry in a vacuum desiccator over phosphorus pentoxide. The residue responds to the U. S. P. identity tests for testosterone.

Assay: (Testosterone) Transfer to a fritted glass funnel of fine porosity an amount of suspension, accurately measured, equivalent to about 0.15 gm. of testosterone. Remove the suspending medium with gentle suction and wash the residue with three 10 ml. portions of water. Quantitatively transfer the testosterone to a 100 ml. volumetric flask by dissolving the residue in 25 ml. of alcohol and washing the funnel thoroughly with five 10 ml. portions of alcohol. Fill the flask to the mark with alcohol, and mix. Transfer to a second 100 ml. volumetric flask 10 ml. of this solution, fill to the mark with alcohol, and mix. Transfer to a third 100 ml. volumetric flask 5 ml. of this last solution, fill to the mark with alcohol, and mix. Spectrophotometrically determine the absorbancy in a 1 cm. quartz cell at 241 mμ, using alcohol as a blank. The concentration of testosterone in the solution in mg./ml. = absorbancy ÷ 54. The amount of testosterone is not less than 90.0 nor more than 110.0% of the labeled amount.

Bio-Intrasol Laboratories, Inc., Brooklyn.

Aqueous Suspension Testosterone with Procaine Hydrochloride 1%: 10 cc. vials. A suspension containing 25 or 50 mg. of testosterone in each cubic centimeter. Preserved with thimerosal 1:10,000.

Lincoln Laboratories, Inc., Decatur, Ill.

Aqueous Suspension Androlin: 10 cc. vials. A suspension containing 25 or 50 mg. of testosterone in each cubic centimeter. Preserved with thimerosal 1:10,000.

Metropolitan Laboratories, Inc., Oyster Bay, N. Y.

Aqueous Suspension Testosterone with Benzyl Alcohol 2%: 10 cc. vials. A suspension containing 10, 25, 50 or 100 mg. of testosterone in each cubic centimeter.

COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION

APPARATUS ACCEPTED

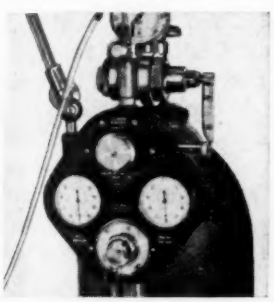
The following additional product has been accepted as conforming to the rules of the Council on Physical Medicine and Rehabilitation of the American Medical Association for inclusion in Apparatus Accepted. A copy of the rules on which the Council bases its action will be sent on application.

RALPH E. DE FOREST, M.D., *Secretary.*

Bennett Pressure Breathing Therapy Unit, Model TV-2P

V. Ray Bennett and Associates, Inc., 320 S. Robertson Blvd., Los Angeles 48, Calif.

The Bennett Pressure Breathing Therapy Unit delivers oxygen under positive pressure to a patient but stops the flow when the pulmonary inflation reaches the point of diminished flow; in other words, it is said to be "flow-sensitive." When it is connected to a patient, a slight inspiratory effort on his part, which creates a very slight initial negative pressure of usually less than 1 cm. H_2O, starts the flow of oxygen into his airways. The pressure then builds up to that set by the control pressure adjustment, and the flow continues at whatever rate the lungs are expanding. The unit will continue to maintain this inspiratory phase until the flow rate is reduced to approximately one or two liters per minute or until the flow direction is reversed. This apparatus therefore differs from the "pressure-sensitive" regulators in which the flow continues until inflation results in a specified back-pressure.



Bennett Pressure Breathing Therapy Unit, Model TV-2P

The essential part about which the apparatus is built is the flow-sensitive Bennett valve, which is to be attached to a cylinder of compressed oxygen. The standard accessories are a mask, mask harness, test lung (a bag of about one liter capacity), nebulizer, wrench, screw driver, cleaning brush, and instruction manual. These are included in the shipping weight, which is 7.7 kg. (17 lb.). A carrying case is optional but the "Flex Arm" to support the connections is a standard part of the unit. Packed for shipment the standard equipment makes a package measuring 26 by 55 by 25 cm. (10½ by 22 by 10 in.).

From sources acceptable to the Council evidence was obtained indicating that the apparatus was well made, that it worked as represented by the manufacturer, and that it was useful in the treatment of certain types of dyspnea and apnea and in the administration of aerosol therapy.

The Council on Physical Medicine and Rehabilitation voted to include the Bennett Pressure Breathing Therapy Unit, Model TV-2P with the stipulation that its use be limited to medical treatments under the direction of a qualified physician.

REPORT OF THE COUNCIL

The Council on Physical Medicine and Rehabilitation has authorized publication of the following article.

AN APPRECIATION

The Council on Physical Medicine and Rehabilitation expresses its gratitude and appreciation for the services of the following consultants, whose assistance in carrying out the work of the Council has been given so freely during 1953: Drs. Thomas D. Allen, Morton Andelman, Thomas L. Ball, Robert W. Benson, Conrad Berens, Theodore B. Bernstein, William Bierman, Edward Bigg, Lester Blum, Ben L. Boynton, Rowine Brown, Raymond Carhart, Mrs. Eva Thompson Carson, Drs. Milton B. Cole, H. G. Davis, Jr., G. deTakats, Donald J. Erickson, David C. Fainer, Alan R. Feinberg, Theodore Friedemann, T. C. Galloway, Clarence J. Gamble, Aram Glorig, Stacy R. Guild, William F. Hamilton, Opal E. Hepler, John S. Hibben, Howard P. House, John W. Huffman, I. F. Hummon, Ernest E. Irons, V. J. Jacey, Richard P. Jahn, Martin H. Kalser, Mary Karp, M. E. Knapp, A. B. C. Knudson, M. M. Kunde, Edward H. Lambert, Ludwig G. Lederer, Alfred Lewy, Edward M. Litin, Douglas Macfarlan, Robert P. MacFate, Raymond W. McNealy, Sedgwick Mead, C. O. Molander, Fred B. Moor, Lester A. Nalefski, Elliot V. Newman, Louis B. Newman, Benjamin H. Orndoff, Mr. H. W. Pavela, Drs. L. W. Peterson, Gerald H. Pratt, James W. Rae, Mr. Luther G. Ramer, Drs. Arthur A. Rodriquez, Max S. Sadove, Oscar A. Sander, D. E. Sanders, Robert S. Schwab, Martin H. Seifert, John K. Sokol, Ralph W. Taraba, Vernon C. Turner, Albert Vanderkloot, H. O. Wernicke, Jessie Wright, and Mr. L. D. Williams.

The following persons have rendered valuable services as members of Advisory Committees:

American Health Resorts.—Drs. F. A. Hellebrandt, M. B. Jarman, Walter S. McClellan, and Euclid M. Smith.

Artificial Limbs.—Drs. Rufus H. Alldredge, Henry H. Kessler, Paul E. Klopsteg, and Eugene E. Record and Messrs. Chester Haddan and J. B. Korrady.

Audiometers and Hearing Aids.—Drs. Richard K. Cook, Kenneth M. Day, Harvey Fletcher, Edmund P. Fowler, Gordon D. Hoople, M. H. Lurie, C. Stewart Nash, and S. Richard Silverman.

Education.—Drs. Robert L. Bennett, Jr., Earl C. Elkins, H. Worley Kendell, Frederic J. Kottke, Donald L. Rose, and Walter M. Solomon.

Electrocardiographs.—Drs. George Fahr, Harold Feil, Harold E. B. Pardee, and William D. Stroud.

Electroencephalography.—Drs. P. Bailey, Edward J. Baldes, Hallowell Davis, Frederic A. Gibbs, Franc D. Ingraham, and Herbert H. Jasper.

Ionizing Radiations.—Drs. Richard Hall Chamberlain, Edwin C. Ernst, E. R. Newell, and Edith H. Quimby and Lauriston S. Taylor.

Ophthalmic Devices.—Drs. David G. Cogan, William F. Hughes, Jr., K. N. Ogle, W. C. Owens, and K. C. Swan.

Respirators.—Drs. Edward L. Compere, Archer S. Gordon, William T. Green, Warren E. Wheeler, James L. Whittenberger, and James L. Wilson.

Sterility and Fertility.—Drs. William J. Dieckmann, Walter J. Meek, and Pendleton Tompkins and Mr. R. R. Olin.

COUNCIL ON FOODS AND NUTRITION

ACCEPTED FOODS

The following product has been accepted as conforming to the rules of the Council.

JAMES R. WILSON, M.D., *Secretary.*

Prince Macaroni Manufacturing Company, Lowell, Mass.

Spinach Pastina

Ingredients: Fancy durum wheat flour, dehydrated spinach, thiamine, riboflavin, niacin, and iron phosphate.

Analysis (submitted by manufacturer).—Total solids 89.5%, moisture 10.5%, ash 1.22%, fat 1.68%, protein (N × 5.7) 12.9%, crude fiber 0.55%, and carbohydrates other than crude fiber (by difference) 73.12%.

Vitamins and Minerals	Mg. per Hundred Grams
Thiamine	0.9
Riboflavin	0.4
Niacin	5.9
Calcium	63
Phosphorus	173
Iron	4.8

Calories.—3.7 per gram; 105 per ounce.

Use.—In the feeding of infants and older children.

THE JOURNAL
OF THE AMERICAN MEDICAL ASSOCIATION
535 N. DEARBORN ST. . . . . CHICAGO 10, ILL.
Editor . . . . . AUSTIN SMITH, M.D.
Associate Editor . . . . . JOHNSON F. HAMMOND, M.D.
Editor for Medical Literature Abstracts . GEORGE HALPERIN, M.D.
Assistant Editor . . . . . WAYNE G. BRANDSTADT, M.D.

Subscription price . . Fifteen dollars per annum in advance
Cable Address . . . . . "Medic, Chicago"

BUILDING THE PROGRAM FOR THE SCIENTIFIC ASSEMBLY

Few members of the American Medical Association who have not had experience in planning programs for large scientific meetings, such as are provided each year at the A. M. A. Annual Meeting in June, are fully cognizant of the time factor essential in the organization of a successful scientific session. Over a long period of years, the Council on Scientific Assembly, which has charge of the program, has evolved with the aid of the Director of Scientific Exhibits, the Secretary of the American Medical Association, and other personnel at the headquarters office in Chicago, a time schedule necessary for the smooth functioning of a somewhat complicated evolutionary process.

Plans for the Annual Meeting usually begin a year in advance, at which time new officers of the various sections are elected or old officers retain their posts. By September the secretary of each section begins to think in terms of the program for the following June meeting, and subjects for consideration are accepted by him from the members of the American Medical Association as early as Labor Day. By mid-October the general outline of a section program is usually well advanced, and at that time the section secretary, and sometimes the section representatives to the Scientific Exhibit, meet in Chicago with the Council on Scientific Assembly and the Director of the Scientific Exhibit and his staff, to consider the program as a whole. Each secretary presents to the Council and to his brother secretaries an outline of his proposed program. At that time, joint meetings are usually arranged and panel discussions or symposiums are given consideration.

More active work on the program takes place between mid-October and mid-December, so that, soon after the first of the year, a secretary ordinarily has his program practically complete. The final arrangements are undertaken during the month of January and the first two weeks of February, the closing date usually being set for February 15. To many members of the American Medical Association this date, four months in advance of the Annual Meeting, seems early, but it is necessary to adhere to it so that the preliminary program can be submitted for publication in THE JOURNAL about April 10. At the same time, the chairman of the Council on Scientific Assembly, with the assistance of the members, makes up the program for the General Scientific Meeting. This ordinarily consists of nine presentations by invitation, the aim being to present to the members of the American Medical Association as a whole a summary of the current advances in medicine by the leaders of the profession. This program too is completed by February 15, so that it may also appear in the Convention Number of THE JOURNAL.

It is frequently desirable to combine a paper presented at a section meeting or before the General Scientific Meeting with an exhibit on the same subject in the Scientific Exhibit. The time relation for applications for space in the Scientific Exhibit is about the same as for papers to be read in the Scientific Assembly. Application forms for requirements must be filed with the Director of Scientific Exhibits at A. M. A. headquarters early in January. Prior to that time, it is also desirable for a member wishing to arrange an exhibit to consult with the section representative, so that exhibits may be coordinated with others on similar subjects and assigned to the appropriate section.

The essential basis for good scientific assembly at an Annual Meeting depends on the coordination between the General Scientific Meeting program; the section program; the Scientific Exhibit; the motion pictures; the presentations associated with exhibits, such as those given in relation to fractures, diabetes, or other aspects of medicine; and more recently the color television program, which has become such an indispensable part of the Scientific Assembly. To coordinate all this requires a continual overseeing the year round by the Council on Scientific Assembly, which also has under its jurisdiction the program for the Clinical Meeting held each December.

The Clinical Meeting presents programming problems of a different type. There are no section meetings and no General Scientific Session similar to those held at the annual meeting. There are, however, Scientific Exhibits, motion pictures, and color television. The program ordinarily consists of a series of clinical presentations to small groups, and the arrangements for its details fall largely on the chairman of the local committee on arrangements and on the chairman of the local program committee. The chairman of the local committee on arrangements, selected locally, is appointed by the Board of Trustees prior to the time of the Clinical Meeting occurring a year in advance of the one in which he is to take charge. This is done so he can attend that meeting and confer with the previous chairman of the local committee on arrangements and with the members of the Council on Scientific Assembly. Thus, from year to year a continuity of effort is established that leads to continued improvement in the character of the Clinical Meeting. Because of the nature of the program and the particular knowledge that the local chairman is likely to have about his own city and the general area where the meeting is to be held, most of the speakers are invited to participate rather than being chosen from applications from the general membership of the American Medical Association. Such applications, however, may be made to the chairman of the local committee on arrangements for the consideration of his program committee. These should be in his hands by May of the year in which the meeting is to be held, so that they can be given considera-

tion when the Council on Scientific Assembly meets with the chairman of the local commitee in that month. The same time schedule, moreover, applies to applications for the Scientific Exhibit.

EMPLOYMENT FOR DIABETICS

The American Diabetes Association has emphasized its philosophy that a controlled diabetic is a good employment risk. Also, by virtue of his capability of performing a full day's work satisfactorily despite his disease, the diabetic should not be classed with the physically handicapped. During the past five years, the association, through its committee on employment, has endeavored to reach some unanimity of opinion in the formulation of recommendations to labor and management regarding the industrial utilization of the diabetic.[1] The structure of the committee is of interest, for extramural members were sought from the United States Civil Service Commission, the United Mine Workers of America, the Industrial Hygiene Foundation, and the diabetes section of the Public Health Service. There was additional contact with the Office of Vocational Rehabilitation of the Federal Security Agency and with the Association of Railroad Engineers.

Mildly diabetic persons, in no need of insulin, present no employment problem. Of persons with severe diabetes, with both insulin and regulated-diet needs, there is the well-controlled and well-regulated patient and the uncooperative and poorly regulated patient. The latter, if hired, would prejudice employers against all diabetics, for they not only are poor work risks because of their diabetes but are potential absentees in the light of complications to come. The well-controlled diabetic has proved his worth and his employment stability in hundreds of organizations employing such persons. Standards suggested by the committee for both employers and prospective workers with diabetes, in essence, are these: 1. A diabetic seeking employment should present a note from his physician stating that he is controlled and undergoes periodic examinations. 2. Diabetics can perform any work for which they are equipped physically, mentally, and educationally. Those taking insulin in large doses should not be assigned to work wherein hypoglycemic attacks would injure either them or others. 3. Diabetics should work straight shifts (unbroken and nonrotating). If a rotating shift is necessary, they should avoid the midnight to 8 a. m. schedule. This is the only concession in terms of hours that a well-controlled diabetic should ask. 4. Certain key personnel in a plant employing diabetics should be familiar with the nature of diabetes and the possibility of coma or insulin reactions. 5. Diabetics should carry cards or tags identifying their condition. 6. The plant physician, in cooperation with the diabetic employee's family physician, should perform periodic blood and urine examinations, if the patient's work schedule precludes visiting his regular laboratory facilities. 7. Each diabetic should have a complete annual physical examination. 8. Transfer or reassignment of a diabetic at the request of the plant physician, when new complications create new risks, is a justified procedure. 9. An insulin-requiring diabetic is considered controlled if his fasting blood sugar is not below normal limits and not over 150 mg. per 100 cc. (Folin-Wu method), if the level after a meal is not higher than 250 mg. per 100 cc., and if he is under regular medical supervision.

It is to be pointed out that excellent liaison has been established by the committee at the national level with the Congress of Industrial Organizations, the American Federation of Labor, and the Railroad Brotherhoods. If necessary this cooperation can be extended downward. With these employment standards in realistic usage by employers and personnel directors, the diabetic applicant should be considered just as eligible for work as the nondiabetic candidate. His effective contribution to industry has been demonstrated, and with continuing cooperative efforts among labor, employers, and professional groups toward hiring the diabetic, employment prejudice will continue to abate.

ILLEGAL OPERATION OF MEDICAL DIATHERMY APPARATUS

The Federal Communications Commission which renders great public service, released recently an interesting and timely public notice, which is of great importance to many physicians. It points out the hazards involved in the illegal operation of medical diathermy apparatus now in use. An abstract of this public notice appears elsewhere (page 1026) in this issue of THE JOURNAL. The commission has set forth the very important reasons for its 1947 ruling, which assigned certain specific frequencies for the exclusive use of medical and industrial diathermy equipment and which gave users of such equipment until June 30, 1952, to acquire apparatus that conformed to the commission's rules. In 1952, the Council on Physical Medicine and Rehabilitation of the American Medical Association petitioned the commission to extend this time limit. The commission did extend the deadline until June 30, 1953, thus giving users a total of six years to comply.

FCC type-approved medical diathermy apparatus manufactured since 1947 is designed to minimize interference with radio communications for home reception, police and fire departments, airlines, airplane landing systems, radio-directed missiles, and television. Excessive radiation from nonconforming diathermy apparatus could be used by enemy aircraft as a navigation aid in flying to target areas in the United States.

Articles in the Council on Physical Medicine and Rehabilitation columns and other articles published in THE JOURNAL since 1947 have called the profession's attention to FCC rules for medical diathermy apparatus. Users of nonconforming medical diathermy apparatus are urged once again to acquire apparatus that can be legally operated. For the information of the profession, the Council on Physical Medicine and Rehabilitation can provide a list of Council-accepted medical diathermy apparatus. This is an extremely important problem requiring the cooperation and understanding of all interested parties, particularly those persons who are using medical diathermy machines not meeting the requirements of the commission.

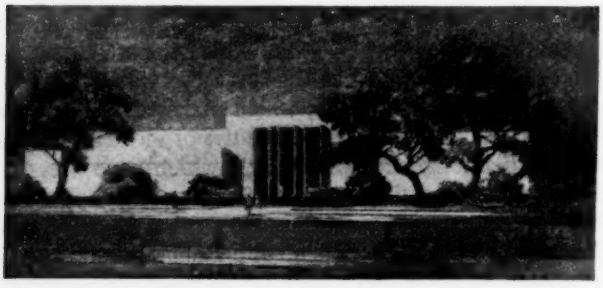
1. Employment of Diabetics: Statement of the Committee on Employment, American Diabetes Association, July, 1952, Diabetes 1: 336 (July-Aug.) 1952. Beardwood, J. T., Jr.: Employment [of Diabetics], ibid. 2: 334 (July-Aug.) 1953.

ORGANIZATION SECTION

TEXAS MEDICAL ASSOCIATION

The Texas Medical Association, which in September, 1952, celebrated its approaching centennial with the dedication of its new memorial library and headquarters building in Austin, was founded Jan. 17, 1853. The 35 physicians who assembled in response to the call "To all regular authorized Physicians of the State of Texas" adopted a constitution and bylaws that included provision for the formation of subordinate societies, the first of which, the Bexar County Medical Society, was chartered in September of that year. After the second assembly of the state association, Nov. 14, 1853, there is an unexplained period in which no meetings were held until 1869 when at a meeting in Houston the body was reorganized as the Texas State Medical Association. In April, 1901, a new charter was issued in the name of the State Medical Association of Texas, a title that was retained until 1951, when the original appellation was resumed.

The headquarters, too, changed frequently through the years, the first building to house offices of the association being the Fort Worth National Bank Building, where headquarters were maintained until 1910. There followed a succession of rented "homes" in the Continental Bank Building, the Western National Bank Building, the Newby Building, and the Medical Arts Building until 1935, when the residence at 1404 West El Paso Street was



Texas Medical Association memorial library and headquarters.

purchased for the central office. In 1948, headquarters were transferred to Austin, where a three story Victorian residence was leased at 700 Guadalupe Street.

The present site at Lamar Boulevard and 19th Street was given to the association by the Travis County Medical Society, and a bequest of $50,000 provided for in an irrevocable trust by Dr. and Mrs. Sam E. Thompson of Kerrville assured the association of the necessary funds for building and operating the library. The auditorium-lounge of the new central office and library building have been named in honor of Dr. Thompson, an emeritus member, who served the state association as president, as member and for 10 years chairman of its board of trustees, and as its delegate to the American Medical Association for 12 years. The Sam Thompson lounge-auditorium, with a seating capacity of 300, in which executive council and other conferences are held, and the library occupy the first floor of the granite-trimmed limestone structure. The library, which has more than 22,000 bound and unbound items, maintains a package service including about 375 periodicals, 7,000 books, 156,000 reprints, 150 professional films, and 50 films suitable for lay audiences. The second floor houses the offices for executive, business, medical services, and public relations activities, as well as those of the state advisory committee, the woman's auxiliary, and the *Texas State Journal of Medicine*, which was founded in July, 1905. Complimentary subscriptions are being provided for senior students of the medical colleges in Texas, at which the association also sponsors lectures and special Medical Students' Days. For graduates of these schools, as well as for other physicians, the association maintains an active placement service, and in furtherance of postgraduate education relays a series of panel discussions by telephone to country medical societies and hospital staffs throughout the state.

The association has long been active in sponsoring medical legislation. In 1907, through the efforts of its legislative committee, the one board medical practice act became a law. In 1917 the vital statistics bill was helped through the legislature, and a more effective workmen's compensation act was passed. Ten years later the association sponsored (1) an amendment to the medical practice act that empowered the board of medical examiners to require the annual registration of all the physicians of Texas and to collect an annual fee of $2 and (2) a bill for reorganization of the state board of health. Still later a minimum standards bill was obtained.

The association has a membership of almost 6,700 physicians. Its woman's auxiliary, which, founded in 1918, was instrumental in establishing an auxiliary in the American Medical Association in 1922, numbers about 4,500 members, whose annual meeting is concurrent with that of the state association. The annual sessions rotate among Dallas, Fort Worth, Galveston, Houston, and San Antonio, where the convention will be held this year May 3-5 under the presidency of Dr. George Turner, El Paso. Other officers of the association include: Dr. Francis J. L. Blasingame, Wharton, president-elect; Dr. Andrew S. Tomb, Victoria, vice-president; Dr. John M. Travis, Jacksonville, secretary; Dr. Thomas H. Thomason, Fort Worth, treasurer; and Mr. N. C. Forrester, executive secretary. The chronicle of the association has been recorded by Dr. Patrick I. Nixon of San Antonio in a 476 page volume "A History of the Texas Medical Association, 1853-1953," which was authorized by the board of trustees in observation of the centennial jubilee.

COUNCIL ON INDUSTRIAL HEALTH

Dr. William P. Shepard, New York, second vice-president, health and welfare division, Metropolitan Life Insurance Company, has been elected chairman of the American Medical Association's Council on Industrial Health. Dr. Shepard succeeds Dr. Anthony J. Lanza, New York, director of the Institute of Industrial Medicine, New York University–Bellevue Medical Center. Two new members of the council also have been elected: Dr. Lemuel C. McGee, Wilmington, Del., medical director, Hercules Powder Company, and Dr. Charles F. Shook, Toledo, Ohio, medical director, Owens-Illinois Glass Company. They succeed Dr. Lanza and Dr. Clarence D. Selby, Port Huron, Mich., whose terms have expired.

The presentation of two American Medical Association citations for distinguished service in the field of industrial health was made at the meeting. The awards went to Dr. Anthony J. Lanza and Dr. Clarence D. Selby, Port Huron, Mich., former medical consultant to the General Motors Corporation, Detroit. Announcement of the citations, the first in the field of industrial health, was made by Dr. George F. Lull, Chicago, Secretary and General Manager of the American Medical Association. Drs. Lanza and Selby have been leaders in the movement for better health conditions among workers.

STUDENTS' ANNUAL CONVENTION MAY 1-3 IN CHICAGO

The Student American Medical Association's fourth annual convention, May 1-3 at the Sherman Hotel, Chicago, will offer a program of panel discussions, speakers, and exhibits. Highlights include a scientific paper, "On Becoming a Physician," by Detroit psychiatrist Leo H. Bartemeier, M.D.; a roundtable discussion entitled, "Tell me, Dean," moderated by John F. Sheehan, M.D., dean of Stritch School of Medicine, Chicago, and a forum, "The Future of Internships." The house of delegates, official policy-making body of the association, will be in session May 1 and 3, with Sunday, May 2, to be devoted to the program proper. Members of SAMA and physicians are cordially invited.

MORE "MARCH OF MEDICINE" TV SHOWS

Of interest to physicians and patients afflicted with arthritis or rheumatism is the second in the spring, 1954, series of "March of Medicine" television programs. Sponsored by Smith, Kline and French Laboratories in cooperation with the American Medical Association, "A Status Report on Arthritis and Rheumatism" will be carried April 29 over the National Broadcasting Company's TV network. The third program in the series, on June 24, will originate from the A. M. A.'s annual meeting in San Francisco. Like last year's series, the new "March of Medicine" programs will be presented at 10 p. m. (EST); it will replace the "Martin Kane" show, which is usually seen at that hour.

AUDIO-DIGEST PROFITS GO TO MEDICAL SCHOOLS

The American Medical Education Foundation recently announced that a new source of funds now is available to medical schools through physician-support of the Audio-Digest Foundation. For a nominal weekly subscription fee, physicians receive from the Audio-Digest Foundation tape-recorded abstracts of current literature and lectures culled from current medical periodicals in all fields of medicine. This foundation, organized by the California Medical Association, will turn over its profits to the AMEF. This should boost the AMEF's 1954 campaign drive for funds from the medical profession to assist the country's 79 approved medical schools.

FEDERAL MEDICAL LEGISLATION

National Fund for Medical Education

Congressman Poff (R., Va.) in H. R. 7914, a companion bill to S. 1748 by the late Senator Taft, would give a federal charter to the National Fund for Medical Education. The Poff measure differs from the Taft bill in only minor details, such as limiting the number of trustees to 17 and requiring that 4 of these be physicians. It specifies also that the contributions can be raised only from private sources. This measure was referred to the Committee on the Judiciary, which has already held a hearing thereon.

STATE MEDICAL LEGISLATION

Arizona

Bills Introduced.—H. 294, proposes regulations for the **compulsory isolation and hospitalization** of persons infected with **tuberculosis.** H. 295, proposes the **creation of a division of tuberculosis control,** the director of which shall be a physician licensed by the state of Arizona who has had not less than five years' experience in the control of tuberculosis. H. 307, proposes the **creation of an Arizona bureau of narcotics enforcement** to coordinate the activities of law enforcement officers and of state agencies in the enforcement of the narcotics act. S. 104, proposes regulations for the creation of **hospital districts.**

Bill Enacted.—S. 42, has become Chapter 6 of the Laws of 1954. It provides that any person who may otherwise validly make a will may, by will or other written, signed, and acknowledged instruments, **dispose of the whole or any part of his body to a teaching institution,** university, college, state department of health, facility designated or maintained by the anatomy board of Arizona, legally licensed hospital, any other agency or commission operating a **tissue, eye, bone, cartilage, or blood bank,** or any other bank of a similar kind designated for the purposes of medical science and the rehabilitation of the maimed.

Kentucky

Bills Introduced.—H. 401, proposes regulations for the effective control of the spread of communicable **tuberculosis.** H. 139, proposes regulations relating to the control of **rabies.** H. 409, proposes the **creation of a department of the handicapped** to exercise all functions of the state relating to the diagnosis, treatment, education, institutional care, vocational guidance, and placement and research for handicapped persons. The commissioner of this department would be required to be a qualified physician licensed to practice in the state with five years' experience in the field of public health administration.

The summary of federal legislation was prepared by the Washington Office of the American Medical Association and the summary of state legislation by the Bureau of Legal Medicine and Legislation.

Massachusetts

Bills Introduced.—H. 2479, proposes to authorize the construction of hospitals for use as homes or **hospitals for aging persons** who are not mentally ill. H. 2495, proposes that in any proceeding to establish paternity the trial court, on motion of the defendant, shall order the mother, her child, and the defendant to submit to one or more **blood grouping tests, to be made by a duly qualified physician** or other duly qualified persons designated by the court, to determine whether the defendant can be excluded as being the father of the child, and the results of such tests shall be admissible in evidence in cases where definite exclusion is established.

Mississippi

Bills Introduced.—H. 615, proposes to require licenses issued by the board of health before any person may engage in selling, distributing, or marketing instruments or **devices designed for** or represented as aiding, improving, or **correcting defective hearing.** H. 634, proposes regulations for the control of **rabies.** H. 656, proposes the **creation of a cancer committee** to serve with the state hospital commission, whose duty it shall be to decide in which cases operating room and/or blood transfusion fees shall be paid and the amount thereof. S. 1517, proposes to require **both applicants for a marriage license to present a medical certificate** indicating that the parties have submitted to a standard serologic test or tests for syphilis.

New York

Bills Introduced.—A.R. 120, proposes to direct the joint legislative committee to inquire into and make a thorough, complete, and careful **study of the admission practices of the medical schools** of the city and state of New York to determine if such admission practice results in discrimination against applicants by reason of race, creed, color, or national origin. A. 2555, proposes the creation of a temporary commission to study the **needs of children and youth** and to recommend a program to meet those needs. A. 2673 and S. 2690, propose the **creation of a board of bioanalytical examiners** and proposes that a person practices analytical technology who holds himself out as being able to perform or who performs or supervises others in performing any bacteriological, biochemical, microscopic, serologic, parasitological, and/or other tests by the practical application of one or more of the fundamental sciences by the use of specialized apparatus, equipment, and methods for the purpose of making scientific investigations, the results of which, when reported to a physician, osteopath, dentist, or podiatrist, may be used as an aid to ascertain the state of health. A. 2792, proposes the creation of a temporary commission to make a study and survey in reference to recommending legislation providing for the **licensing of all those except medical doctors who prescribe or sell hearing aids.** A. 2800, to amend the education law, proposes to define the word "physician" as including a practitioner of either **osteopathy or naturopathy.** A. 2832, to amend the social welfare law, proposes that no certificate of incorporation shall be filed that includes among its corporate purposes the operation of **private proprietary hospitals,** nursing homes, infirmaries, homes or institutions for convalescents, aged, or indigent persons, and lying-in homes operated for profit **except with the written approval of the board and a justice of the supreme court.** A. 3040 and S. 2452, propose the **creation of a state board of massotherapists examiners** and defines the practice of massage as a scientific system of applied activity of the muscular structures of the human body, by the means of stroking, kneading, tapping, and vibrating of such structures with the hands for the cardinal purpose of improving the circulation. It excludes the reduction of fractures or dislocations of the bones. A. 2856 and S. 2505, propose that every **hospital** supported in whole or in part by public funds or the solicitation of private subscriptions and to which patients are submitted on payment of a charge or fee or at which persons receive medical and/or surgical treatment on payment of a charge or fee **may employ physicians and surgeons under a contract or salary arrangement for the medical diagnosis and/or treatment of patients only when such diagnosis and treatment is a public charge.** In all other cases such medical diagnosis and/or treatment shall be rendered to patients in such hospital independently of other hospital charges and under contractual relationship between the patient and the physician. A. 2973, to amend the traffic law relating to the **withdrawal of blood for the purpose of determining the alcoholic content** therein, proposes that physicians shall not be liable for damages in any civil suit for withdrawing such blood except for gross negligence in the performance of such withdrawal. S. 2694, to amend the public health law, proposes circumstances under which a **city health department may secure qualified full-time physicians for service during an emergency,** even though such physicians are not citizens of the United States.

Rhode Island

Bill Introduced.—H. 748, proposes that no **general release or settlement** in writing signed by a person confined in a hospital shall be admissible in evidence in a trial or action to recover damages for personal injuries necessitating the hospitalization if the statement was obtained within fifteen days after the injuries except under certain specified circumstances.

Virginia

Bill Enacted.—S.J.R. 18, was adopted Feb. 10, 1954. It directs the Virginia Advisory Legislative Council to study the **desirability and feasibility of providing specially designed geriatric facilities** for the care of aged patients.

MEDICAL NEWS

CALIFORNIA

Medical Tour of Europe.—The California Medical Association has arranged its first annual grand medical tour of Europe, April 5-May 5. The itinerary provides for about one week in France and one in Italy, three days in England, three in Holland, two in Switzerland, and one in Belgium. Medical meetings are scheduled for Paris, Rome, Lucerne, and London. The total cost of the tour is $1,788 (plus $45.29 tax). Members' wives and children under 22 will receive the benefit of a family plan reduction. The party will leave San Francisco or Los Angeles by United Air Lines DC-6B Mainliner, April 5, arriving in New York the same evening and leaving the following day for Paris. On the return trip, arrival is scheduled for New York at noon, May 4, and departure for California the following day. Information may be obtained from the California Medical Association, 450 Sutter St., San Francisco 8.

ILLINOIS

Dr. Windle Goes to Maryland.—William F. Windle, Ph.D., scientific director of the Baxter Laboratories, Morton Grove, has been appointed chief of the Laboratory of Neuroanatomical Sciences of the National Institute of Neurological Diseases and Blindness, where he will continue his investigations into the problems of nerve regeneration. Dr. Windle previously served as chairman of the department of anatomy of the University of Pennsylvania, Philadelphia, and at the University of Washington, Seattle, and as director of the Institute of Neurology at Northwestern University, Chicago. He has held the Harvey lectureship, the War-Navy award, and the Sesquicentennial medal of the University of Louisville.

Chicago

Personal.—At the meeting of the Society of Illinois Bacteriologists, Feb. 27, Dr. Tibor Benedek, assistant clinical professor of dermatology and syphilology, Stritch School of Medicine of Loyola University, delivered a paper on the Bacillus endoparasiticus Benedek 1927.——Dr. Benjamin M. Gasul will present before the Memphis Heart Association "Advances in Pediatric Cardiology" March 22 and will hold a clinic the following morning.

Northwestern's New Medical Research Building.—According to J. Roscoe Miller, M.D., president of Northwestern University, construction of the seven-story Morton Medical Research Building was begun in February at 310 E. Superior St. on the Chicago campus. The new building will be financed by a 2 million dollar bequest left to the university by Mrs. Margaret Gray Morton, and it will be connected with a wing of the Ward building. A special feature will be movable metal wall partitions that can be set up at any 10-foot interval within the building, allowing laboratories to be converted at a moment's notice to almost any size desired. Fourteen specially designed chemical hoods for experiments with noxious fumes will be built. An electrical power supply of 576,000 watts will be conducted in a bus duct system unusual in a research building, and a continuous run of copper bars for conducting electricity will be constructed in all corridors of the building. Switches can be attached at any 1-foot interval along the bars to provide power at almost any point in the building.

KANSAS

Clendening Lectures.—Dr. Douglas Guthrie, lecturer in the history of medicine at the University of Edinburgh in Scotland, will be the Clendening memorial lecturer March 24-25. On Wednesday, 11 a. m., at the University of Kansas School of Medicine, Lawrence, his topic will be "Witchcraft and Witch Doctors." In the evening Dr. Guthrie will speak in the Library of the History of Medicine at 8 p. m. The lecture at the University of Kansas School of Medicine, Kansas City, at 12 noon the following day will be "Lister and His Achievement." Physicians and students are invited.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

MAINE

Clinics to Be Held.—Pediatric clinics are scheduled to be held at the Northern Maine Sanatorium, Presque Isle, March 24, 1:30 p. m., and at the Eastern Maine General Hospital in Bangor March 26, 1:30 p. m. A tumor clinic has been scheduled at the latter hospital March 25, 1:30-3 p. m., and a cardiac clinic, March 26, 9 a. m. to 12 noon.

Society News.—At a recent meeting of the Maine Society of Anesthesiologists the following officers were elected: Dr. George E. Sullivan, Bingham, president; Dr. Valentine J. Moore, Waterville, vice-president; and Dr. John R. Lincoln, Portland, secretary-treasurer.——The Maine Society of Radiologists recently elected the following officers: Dr. John Southworth, Togus, president; Dr. Clark F. Miller, Lewiston, vice-president; Dr. Jack Spencer, Portland, secretary-treasurer; and Dr. Forrest B. Aimes, Bangor, councilor to the American College of Radiology.

MARYLAND

Cancer Lecture.—Under the sponsorship of the house staff of Sinai Hospital, Baltimore, Dr. Sidney Farber, professor of pathology, Harvard Medical School, Boston, will deliver the Dr. Jack W. Kolson memorial lecture, "Approaches to the Chemotherapy of Cancer," March 25, 8:30 p. m. in Hurd Hall, Johns Hopkins Hospital, Baltimore. Physicians, medical students, and those in allied fields are invited.

MASSACHUSETTS

Dr. Rhoads to Lecture on Neoplastic Diseases.—The Alpha Theta chapter of the Phi Delta Epsilon fraternity at Tufts College Medical School, Boston, will hold the seventh annual lectureship March 22. The guest speaker will be Dr. Cornelius P. Rhoads, Sloan Kettering Institute, Memorial Center for Cancer and Allied Diseases, New York, who will discuss "Advances in the Treatment of Neoplastic Diseases."

Symposium on Poliomyelitis.—Dr. Charles A. Janeway, Boston, will serve as moderator for the following program of the Norfolk District Medical Society, March 23, 8:15 p. m. in the Jimmy Fund Building Auditorium, Children's Medical Center, 35 Binney St., Boston:

Louis Weinstein, Boston, Diagnosis of Acute Poliomyelitis.
R. Cannon Eley, Boston, Treatment of the Usual Case.
Bertrand C. Kriete, Boston, Recognition and Management of Dangerous Complications of the Acute Disease.
Thomas H. Weller, Boston, Poliomyelitis Viruses and Vaccines.
Roy F. Feemster, Boston, Present Status of Methods of Prevention and Control.

Course on Measurement of Pulmonary Function.—A course in the measurement of pulmonary function in health and disease will be held for physicians in the New England area at Boston City Hospital, March 22-26 under the sponsorship of the American Trudeau Society, in cooperation with the Massachusetts Tuberculosis and Health League, Harvard Medical School, Boston University School of Medicine, and Tufts College Medical School. The course will present the fundamental concepts and methods of analysis of normal and abnormal physiology of the lungs in a manner that can be readily understood by physicians unfamiliar with this type of chest work. Information may be obtained from Dr. Edward J. Welch, chairman, ATS Regional Committee on Postgraduate Courses, 1101 Beacon St., Brookline 46.

MINNESOTA

George E. Fahr Lecture.—The annual George E. Fahr lecture, "Some Pitfalls in the Care of Cardiacs," will be given March 23 in Owre Amphitheater on the University of Minnesota campus in Minneapolis by Dr. Samuel A. Levine, clinical professor, Harvard Medical School, Boston. Dr. Levine will also participate in the continuation course in cardiovascular diseases which will be held March 22 to 24 at the Center for Continuation Study.

Dr. Ulrich Honored.—The Hennepin County Tuberculosis Association, which on Dec. 2, 1953, observed the half-century mark in its "building of the wall" against tuberculosis, presented to Dr. Henry L. Ulrich, Minneapolis, a plaque in recognition of his many years of work toward the eradication of tuberculosis. Speakers at the golden jubilee of the association, which was founded a year before the creation of the National Tuberculosis Association and two years before the Minnesota state association, included Drs. S. Marx White, Walter J. Marcley, and Stewart C. Thomson, Minneapolis.

NEW JERSEY

Medicolegal Meeting.—The New Jersey Neuropsychiatric Association will hold an evening medicolegal meeting at the Academy of Medicine, Newark, March 24. The Briggs law and its relationship to psychiatric testimony will be discussed. Members of the medical and legal professions are invited.

Personal.—Dr. Samuel Cohen was recently appointed director of medicine at the Berthold S. Pollak Hospital for Chest Diseases, Jersey City.——Dr. Richard H. Roberts, Summit, has assumed the position, newly created by Ciba Pharmaceutical Products, of senior clinical research associate on the West Coast, where his address is 417 Market St., San Francisco.

NEW YORK

Cancer Program.—A cancer teaching day will be held March 25, 9 a. m. to 5 p. m., at Albany Medical College under the sponsorship of the college, the Medical Society of the County of Albany, Medical Society of the State of New York, and the New York State Department of Health. The program, conducted by members of the Jefferson Medical College of Philadelphia, will be presented in two parts. In the morning "Cancer of the Bronchus" will be discussed by Drs. John H. Gibbon Jr., Peter A. Herbut, Louis H. Clerf, and Theodore P. Eberhard, and in the afternoon "Treatment of Leukemia" will be presented by Drs. Franklin R. Miller, John S. Flint, and Theodore P. Eberhard.

Buffalo Clinical Day.—The Alumni Association of the University of Buffalo School of Medicine will conduct its annual Clinical Day, March 20, at the Hotel Statler, Buffalo. Physicians are invited to attend the sessions and join in the luncheon-get-together on the same payment basis as Alumni Association members. Dr. George M. Masotti, president of the alumni association, announces the following program:

Problems in the Resuscitation of the Newborn Infant, Richard L. Day, New York.
Surgical Treatment of Ulcerative Colitis—A Medical and Surgical Program, Garnet W. Ault, Washington, D. C.
Portacaval Shunting for Portal Hypertension, with Special Reference to Cirrhosis of the Liver, Arthur H. Blakemore, New York.
Early Diagnosis of Malignant and Pre-Malignant Lesions of the Colon and Rectum, Neil W. Swinton, Boston.
Some Pitfalls in the Care of Cardiacs, Samuel A. Levine, Boston.
Measures in the Prevention of Maternal Morbidity and Mortality, Robert A. Ross, Chapel Hill, N. C.

New York City

Society News.—The Association for the Aid of Crippled Children has moved to 345 E. 46th St. (OXford 7-3150).——At the meeting of the Society for the Study of Blood, March 23, 8:30 p. m., at the New York Academy of Medicine, "Platelet Transfusions in the Control of Thrombocytopenic Bleeding" will be discussed by Dr. Allen H. Minor, research associate, Lennox Hill Hospital, and "Immunologic Mechanisms in Idiopathic Thrombocytopenic Purpura" by Dr. William J. Harrington, senior assistant surgeon, U. S. Public Health Service, National Institute of Arthritis and Metabolic Diseases, Bethesda, Md.

Lectures on Anesthesiology.—Modern scientific method and modern statistics with special application to anesthesiology will be presented by Dr. Philip H. Sechzer, director of anesthesiology, Fordham and Seton hospitals, and consultant, U. S. Public Health Service, Staten Island, at 1:30 p. m. March 25, April 22, and May 20 in Ward 9, auditorium, Fordham Hospital. The topics to be presented are: logic and scientific method, the meaning of statistics, how to evaluate evidence, bias and variations, enumeration data, confidence limits, chi square test, mensuration data, T test, correlation and regression, simple experiments, and factorial design. The profession is invited.

Biology Seminars.—New York University is presenting a series of graduate biology seminars open to all interested persons, at 5 p. m. on Friday evenings in NYU's Main Building, Washington Square Center. On March 19, Dr. Albert Tannenbaum, director, department of cancer research, Medical Research Institute of Michael Reese Hospital, Chicago, discussed "Nutrition in Relation to the Cancer Process," and, on March 26, George Wald, Ph.D., professor of biology, Harvard University, Cambridge, Mass., will present "The Molecular Basis of Vision." The following programs complete the schedule: April 2, John Tyler Bonner, Ph.D., associate professor of biology, Princeton University, Princeton, N. J., "Recent Studies on the Development of the Slime Mold, Dictyostelium Discoideum"; April 23, Horace W. Stunkard, Ph.D., professor of biology at New York University, "Parasitism as a Biological Phenomenon"; and April 30, James H. McGregor, Ph.D., professor emeritus of zoology at Columbia University, "Comparison of Apes and Man."

OREGON

Harmon Lectures.—The Harmon lecture series, presented annually by the Library Association of Portland, will be co-sponsored this year by the University of Oregon Medical School, Portland, making possible medical talks designed for laymen. The lectures, presided over by Dr. Daniel H. Labby, associate professor of medicine, will include: Dr. Howard P. Lewis, professor and head of the department of medicine, "Facing the Medical Problems of Aging," March 24; Dr. S. Gorham Babson, assistant clinical professor of pediatrics, "The Signs of Emotional Strain in Our Children," March 29; Dr. Roy L. Swank, professor of neurology, "Approaches to a Mysterious Disease—Multiple Sclerosis," April 8; and Dr. Joseph F. Paquet, clinical associate in medicine, "What's New in Poliomyelitis," April 21.

PENNSYLVANIA

Memorial to Dr. Laplace.—The Aid Association of the Philadelphia County Medical Society announces the establishment of the Dr. Louis B. Laplace Memorial Fund as a tribute to Dr. Laplace who died a few days before the termination of his service as a director of the association. Individual or group contributions may be sent to the Aid Association, Philadelphia County Medical Society, 21st and Spruce streets, Philadelphia 3, earmarked for the "Dr. Louis B. Laplace Memorial Fund."

Philadelphia

Alpha Omega Alpha Lectureship.—The Temple University chapter of Alpha Omega Alpha will present its annual lectureship March 22, 4 p. m., in the auditorium of Temple University School of Medicine, 3400 N. Broad St. Dr. Charles A. Hufnagel, professor of surgical research, Georgetown University School of Medicine, Washington, D. C., will discuss "The Reconstructive Concept in the Treatment of Diseases of Major Arteries."

VERMONT

Surgeons Meet in Montpelier.—The spring meeting of the section on surgery of the Vermont State Medical Society will be held March 25 at the Pavilion in Montpelier. Dr. Robert R. Linton, assistant professor of clinical surgery, Harvard Medical School, Boston, will discuss "New Parts for Old in the Surgical Treatment of Diseases of the Major Arteries" before the social hour which precedes the 6:30 banquet. He will also show his film "Treatment of Venous Thrombosis of the Lower Extremity." Dinner reservations are essential. Dr. Benjamin F. Clark, St. Johnsbury, is chairman of the program committee.

VIRGINIA

Postgraduate Day Program at Roanoke.—The fifth annual postgraduate day program of the Memorial and Crippled Children's Hospital, Roanoke, will be held at the Hotel Roanoke, March 24. Dr. Alto E. Feller, University of Virginia School of Medicine, Charlottesville, will serve as moderator for a panel discussion "Uses and Complications of Therapeutic Agents for Infections." He will also take part in a clinicopathological conference with Dr. James C. Gale, Roanoke. Dr. Keith S. Grimson, Duke University School of Medicine, Durham, N. C., will present "Medical and Surgical Management of Peripheral Vascular Disease." Dr. Edward C. Curnen, University of North Carolina School of Medicine, Chapel Hill, will discuss "Immunization Against Poliomyelitis," and Dr. Thomas D. Moore, University of Tennessee College of Medicine, Memphis, "Recognition and Management of Ureteral Injuries."

Symposium on Pulmonary Disease.—The Medical College of Virginia, Richmond, will present the Stoneburner lectures March 24-26. Dr. Alvan L. Barach, clinical professor of medicine, Columbia University College of Physicians and Surgeons, New York, will give the opening address "Management of Pulmonary Emphysema," Wednesday, 8:30 p. m., and will speak at the same time on Thursday, on "Mechanically Induced Bronchial Drainage in Respiratory Disease. The Principles and Techniques of Exsufflation with Negative Pressure." The Thursday session will open at 10 a. m. with a discussion on anatomy of the lung by Louise L. Jones, assistant professor of anatomy, Medical College of Virginia. Dr. Herbert C. Maier, assistant clinical professor of surgery, Columbia University College of Physicians and Surgeons, will discuss "Selection of Patients for Pulmonary Surgery" and "Diagnosis and Treatment of Spontaneous Pneumothorax." Dr. Averill A. Liebow, professor of pathology, Yale University School of Medicine, New Haven, will present the pathology of emphysema and (Friday) pulmonary circulation. "Practical Aspects of Pulmonary Function" will be the topic Thursday of Dr. George W. Wright, St. Luke's Hospital, Cleveland, who on Friday will discuss "The Effect of Collapse and Surgical Measures on Pulmonary Function." Dr. Osler A. Abbott, clinical assistant professor of surgery, Emory University School of Medicine, Emory University, Ga., will present "The Treatment of Lung Abscess" on Thursday and Friday "The Treatment of Bronchiectasis." The program will also include the following presentations by Richmond physicians:

Count D. Gibson Jr., Treatment of Pneumonia.
John L. Patterson Jr., Clinical Significance of Dyspnea.
Edward S. Ray, Pulmonary Osteoarthropathy.
F. Philip Coleman, Carcinoma of the Lung.
Lewis H. Bosher Jr., Pre- and Post-Operative Care in Thoracotomy.
Alfred M. Decker, Surgery in Pulmonary Tuberculosis.

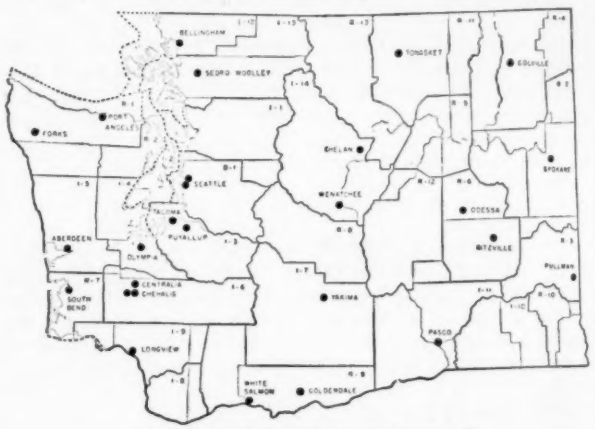
WASHINGTON

Hospitals Built with Hill-Burton Aid.—Hospital construction in the state of Washington has received a great stimulus through the federal Hospital Survey and Construction Act (Hill-Burton), which became effective on Aug. 13, 1946. This act established a basis for the allocation of federal funds to assist local communities throughout the nation in building hospitals and health facilities. The federal funds are allocated each year, each state being required to conduct continuous research and investigation of hospital needs within the state. These data are revised annually to show the changing needs of the state, and on this information a state plan is developed. The first Washington state hospital plan, completed in 1947, after a survey of existing facilities and analysis of the needs of the state, recommended the construction of hospitals, which, when built, would, together with existing hospitals, provide physical facilities to insure that every citizen may receive the maximum benefit that medical science has to offer. Special consideration is given to shifts in the population, usage of existing facilities, and such economic and sociologic factors as pertain to sound hospital development.

Since 1947 the Hill-Burton hospital construction program has aided in 34 projects throughout Washington, of which 25 are completed, 5 are under construction, and 4 are near the construction stage. The number of hospital beds thus added to the state facilities for the care of the sick varies in the individual projects from 13 to 507, some of the larger numbers being the total capacity after an addition is completed to an existing facility. A few of the new facilities are shown on the opposite page.

Since 1947, when the program was begun, more than 30 million dollars worth of hospital and health center facilities were made possible, of which sum a total of $6,500,000 in federal aid had been allocated to approved projects. Assistance to local communities was given in amounts up to 40% of the cost of construction.

The Washington State Department of Health published a brochure comprising a progress report on the Hill-Burton program, 1947-1952, which shows that the first Hill-Burton hospital project to be completed as well as the first hospital to be built by a hospital district in the state was the Klickitat Valley General Hospital at Goldendale, which is typical of community efforts to provide modern hospital service in isolated rural areas. This new hospital is owned and operated by County Hospital District no. 1, which was formed by the almost unanimous vote of the local population. It is a single-story facility with an initial capacity of 23 beds, serving a farming and lumbering community of about 8,000 persons. Specialists in orthopedic surgery, gynecology, and radiology, come in from The Dalles, 32 miles southwest and across the Columbia River by ferry, from Yakima, 81 miles north, and from Portland, thus making special as well as general services available to the community.



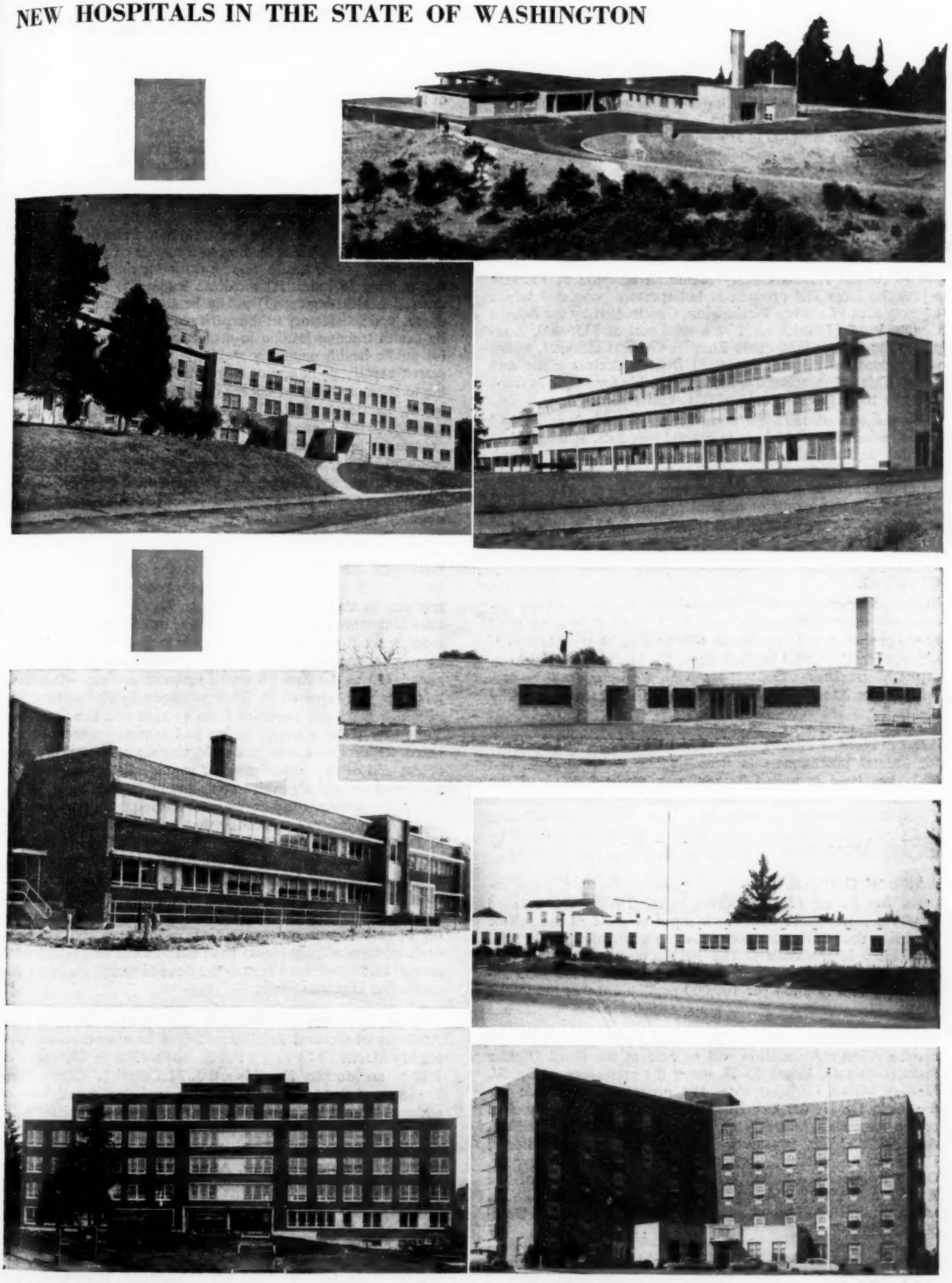
• Hill-Burton Construction Projects, 1952
—Hospital Service Area Boundaries

The new 121 bed St. Joseph Hospital at Aberdeen replaces its 30-year-old predecessor situated on the same grounds nearby. Aberdeen is a logging and seaport city located on Grays Harbor in an area having a high priority on Hill-Burton aid under the state hospital program. With 50,000 persons the area has 4.5 general hospital beds per thousand population in use as compared with a state average (at the time the brochure was published) of 3.6 beds per thousand. The higher average usage is due mainly to the large percentage of the population in this area covered by prepaid medical service plans and to the extreme hazards of the forest industries, in which about 42% of the working population is employed. With the help of the state department of health, funds made available through the Hill-Burton program, together with funds from a bequest, the Sisters of the Convent of St. Rose were encouraged to go ahead with an entirely new hospital at a cost no greater than it had been estimated to construct an addition to the old building. The new hospital, which cost $1,483,000, is the largest of the three general hospitals serving the Aberdeen hospital service area.

The new Yakima Valley Memorial Hospital of 160 beds has a 23 bed psychiatric unit, which is the first such unit in the state to receive assistance from the Hill-Burton program. The entire facility was built and equipped by the Yakima Valley Memorial Hospital Association at a cost of $2,413,000, toward which sum a federal grant was necessary to enable completion of the psychiatric unit and equipping the hospital.

The Lutheran Minor Hospital for the Treatment of Chronic Illness is the first new hospital in the state to deal specifically with this problem. Located at Puyallup, adjacent to the Lutheran Home for the Aged and closely affiliated with the local general hospital, it has 76 beds and facilities for physical and occupa-

NEW HOSPITALS IN THE STATE OF WASHINGTON



Reading from top and left to right are Skyline Hospital, White Salmon; St. Helen Hospital, Chehalis; Olympic Memorial Hospital, Port Angeles; Adams County Memorial Hospital, Ritzville; St. Martin's Hospital, Tonasket; Memorial Hospital, Sedro Woolley; St. Joseph Hospital, Aberdeen; and Yakima Valley Memorial Hospital, Yakima.

tional therapy, minor surgery, and laboratory and x-ray. The Lutheran Welfare Society undertook to construct this hospital with funds raised through public subscription and mortgage loan, but, as these were insufficient, a Hill-Burton grant was approved for the purchase of equipment.

University, state, and Hill-Burton funds made possible construction of the first unit of the 300 bed University of Washington Teaching Hospital in Seattle, a clinic research unit adjoining the Health Sciences Building on the university campus. Physicians throughout the state will be able to refer patients to the outpatient clinic in this unit to receive the benefit of highly specialized studies and examinations. This structure contains a small referral diagnostic clinic for outpatients and 24 beds for inpatients, which beds will be limited to medical research purposes.

The 20 bed Adams County Memorial Hospital at Ritzville serves the large and prosperous but sparsely populated wheat farming area of eastern Washington. Constructed by the Adams County Public District no. 1 at a total cost of $336,000, it replaces the former inadequate Ritzville General Hospital, which was housed in quarters converted from a doctors clinic and home. This new hospital has made it possible for surgeons from Spokane to serve the people of this rural area.

By the end of 1952 the construction of new facilities had added more than 1,000 beds in general hospitals, at which time the state had a total of 8,313 general hospital beds. The previously mentioned brochure states that at that time a leveling off in the need for general hospital beds appeared to have taken place partly as a result of a trend toward a shorter length of patients' stay in the hospital and "partly due to the new 'wonder' drugs that have made some use of hospitals unnecessary."

GENERAL

Eastern Surgeons Meet in Boston.—The Eastern Surgical Society will meet in Boston, March 26-27. From 8:30 a. m. until noon, operative and dry clinics will be held at the Massachusetts General Hospital on both days. On Friday after luncheon at the Massachusetts General Hospital, a program will be presented from 2 to 4 p. m. at the Peter Bent Brigham Hospital by the staff.

Areas in Need of Physicians.—Information concerning openings for general practitioners in Pennsylvania, West Virginia, and Ohio has been compiled by the area medical office of the United Mine Workers of America Welfare and Retirement Fund, Empire Building, 507 Liberty Ave., Pittsburgh. Information concerning openings in other states has been compiled by the national office and other area medical offices of the fund.

Accidental Deaths in 1953.—The National Safety Council reports that during 1953, accidents in the United States killed 95,000 persons (more than three times as great as the toll of American dead during the entire Korean war), injured 9,600,000 and cost 9 billion dollars. The 1953 accident death toll was, however, 1,000 below that of the year before, and the 1953 all-accident death rate (60 per 100,000 population) was the lowest on record.

Allergists Meet in Atlanta.—The annual meeting of the Southeastern Allergy Association will be held at the Hotel Dinkler Plaza in Atlanta, March 25-27, under the presidency of Dr. W. Lindsay Miller, Gadsden, Ala. The program will include addresses by Dr. Homer E. Prince, Houston, Texas, president-elect of the American Academy of Allergy; Dr. John M. Sheldon, Ann Arbor, Mich., president, American Academy of Allergy; Dr. Ethan Allan Brown, Boston, editor, *Annals of Allergy;* Dr. Oscar Swineford, Charlottesville, Va., past president of the American Academy of Allergy; and Albert S. Richardson, Ph.D., associate director of the chemical division of Proctor and Gamble, Cincinnati.

Symposium on Drug Resistance.—A symposium on the origins of resistance to drugs will be presented under the sponsorship of the Office of Naval Research and the University of Pennsylvania March 25-27 in Washington, D. C. The symposium will consist of six successive sessions, each devoted to one of the following topics: (1) origins of microbial drug resistance; (2) tolerance and addiction to drugs, and alcoholism; (3) resistance to insecticides, herbicides; (4) factors in resistance to infectious agents; (5) biochemical mechanism of carcinogenesis and the nature of tumor immunity; and (6) integration of the questions of resistance from the genetic, physiological, and chemical points of view.

Scholarships in Public Health.—The Harvard School of Public Health is initiating postgraduate scholarships in amounts ranging up to $5,000 to qualified candidates desiring to study at the school during the academic year 1954-1955. These scholarships are available to (1) physicians, dentists, and veterinarians interested in preventive medicine and seeking training in one or more public health specialties leading to a master of public health or a doctor of public health degree; (2) industrial physicians seeking training in industrial medicine leading to a master of industrial health degree; (3) public health nurses with a college degree and satisfactory field experience who wish additional public health training leading to a master of public health degree; (4) public health engineers seeking additional training and research experience in one or more public health specialties leading to either a master of science in hygiene or a doctor of science in hygiene degree; and (5) college graduates with academic experience in the natural sciences who desire training and research experience in one of the sciences related to public health and leading to either a master of science in hygiene or a doctor of science in hygiene degree. Applications must be filed by April 30. Winners will be announced June 1. Information may be obtained from the Secretary, Harvard School of Public Health, 55 Shattuck St., Boston 15.

Psittacosis Reported.—According to the U. S. Public Health Service, psittacosis virus was recently recovered from two parrots sent to a game farm in Virginia. X-ray examination of the attendant who cared for the birds showed evidence of pneumonitis about one week after discharge from a hospital where he was treated for pneumonia. A specimen of blood from the attendant was positive 1:8 with Lygranum antigen.—Psittacosis infection was reported in three parakeets in Connecticut, one of which had been imported from Florida and two from New York City from a source which had previously been incriminated. Reports have been made of four suspected cases in human beings and of two actual cases of psittacosis, in both of which the blood showed a positive complement fixation test in a titer of 1:128.—Pursuant to information received from the Tennessee health commissioner, indicating that human cases of psittacosis in that state had been traced to contact with parakeets purchased in North Carolina, an investigation by the North Carolina State Board of Health disclosed six cases of atypical pneumonia in the family of the owner of the aviary from which the parakeets had been purchased. It was reported that a large number of persons raise psittacine birds in this area and that many cases of atypical pneumonia have occurred. Blood samples and several birds have been sent to the virus laboratory of the Communicable Disease Center.

General Practitioners Meet in Cleveland.—The American Academy of General Practice will hold its annual scientific assembly March 22-25 at the Public Auditorium in Cleveland. At 1:30 p. m. Monday Dr. Edward J. McCormick, Toledo, Ohio, President of the American Medical Association, will deliver the address "The Physician as a Citizen." Presentations by Dr. Roscoe L. Pullen, Columbia, Mo. (Why Train for General Practice?) and Dr. Joseph Hughes, Philadelphia (The Family Doctor and Our Youth) will follow. At 4 p. m. Dr. Howard A. Rusk, New York, will conduct a clinic on rehabilitation. The Tuesday session will open at 9 a. m. with presentation of "Significance of Thyroid Enlargement" by Dr. George Crile Jr., Cleveland, which will be followed by "Emergency Room Surgery" by Dr. Donald M. Glover, Cleveland, "Allergic Dermatitis" by Dr. George H. Curtis, Cleveland, and "Commonly Missed Diagnoses of Skin Diseases" by Dr. Clarence S. Livingood, Detroit. The afternoon session will be devoted to a symposium on peptic ulcer (Dr. C. Stuart Welch, Albany, N. Y., moderator) and a

symposium on tuberculosis (Dr. Sol Katz, Washington, D. C., moderator). Wednesday at 11:30 a. m. Dr. Richard W. TeLinde, Baltimore, will discuss "Unnecessary Surgery in Ob-Gyn." His presentation will be preceded by "Medical Complications in Obstetrics" by Dr. Robert M. Myers, Kansas City, Mo., "Management of Vaginal Bleeding" by Dr. Robert L. Faulkner, Cleveland, and "The Routine Multip" by Dr. Allan C. Barnes, Columbus, Ohio. The Wednesday afternoon session will open with a symposium "Headache, Dizziness and Syncope," which will be moderated by Dr. Theodore J. von Storch, New York. At 4 p. m. Sir Alexander Fleming will have as his topic "Beginning of Antibiotic Therapy." The afternoon session will conclude with a symposium on antibiotics in infectious diseases, for which Dr. Paul A. Bunn, Syracuse, N. Y., will serve as moderator. On Thursday rheumatic arthritis will be discussed by Dr. Thomas F. Frawley, Albany, N. Y., other collagen diseases by Dr. Robert Strecher, management of the anemias by Dr. William S. Middleton, Madison, Wis., and recent advances in therapeutics by John C. Krantz Jr., Ph.D., Baltimore. The president's reception will be held in the ballroom of the Hotel Cleveland Wednesday evening.

MEETINGS

AMERICAN MEDICAL ASSOCIATION: Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.
1954 Annual Meeting, San Francisco, June 21-25.
1954 Clinical Meeting, Miami, Florida, Nov. 29-Dec. 2.
1955 Annual Meeting, Atlantic City, N. J., June 6-10.
1955 Clinical Meeting, Boston, Nov. 29-Dec. 2.
1956 Annual Meeting, Chicago, June 11-15.

AERO MEDICAL ASSOCIATION, Hotel Statler, Washington, D. C., March 29-31. Dr. Thomas H. Sutherland, P. O. Box 26, Marion, Ohio, Secretary.

ALABAMA, MEDICAL ASSOCIATION OF THE STATE OF, Mobile, April 15-17. Dr. Douglas L. Cannon, 537 Dexter Ave., Montgomery, Secretary.

AMERICAN ACADEMY OF GENERAL PRACTICE, Cleveland, March 22-25. Mr. Mac F. Cahal, 406 West 34th St., Kansas City 2, Mo., Executive Secretary.

AMERICAN ACADEMY OF NEUROLOGY, Shoreham Hotel, Washington, D. C., April 29-May 1. Dr. Alexander T. Ross, 1040 West Michigan St., Indianapolis 7, Secretary.

AMERICAN ASSOCIATION OF ANATOMISTS, Hotel Galvez, Galveston, Texas, April 7-9. Dr. Normand L. Hoerr, 2109 Adelbert Road, Cleveland 6, Secretary.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION, Webster Hall Hotel, Pittsburgh, May 14-15. Dr. Jack Matthews, 1617 Cathedral of Learning, University of Pittsburgh, Pittsburgh 13, Secretary.

AMERICAN ASSOCIATION OF THE HISTORY OF MEDICINE, Hotel Taft, New Haven, Conn., May 6-8. Dr. Samuel X. Radbill, 7043 Elmwood Ave., Philadelphia 42, Secretary.

AMERICAN ASSOCIATION OF IMMUNOLOGISTS, Ambassador Hotel, Atlantic City, N. J., April 11-15. Dr. John Y. Sugg, 1300 York Ave., New York, Secretary.

AMERICAN ASSOCIATION OF PATHOLOGISTS AND BACTERIOLOGISTS, Philadelphia, Pa., April 8-10. Dr. Alan R. Moritz, 2085 Adelbert Road, Cleveland 6, Secretary.

AMERICAN ASSOCIATION OF RAILWAY SURGEONS, Drake Hotel, Chicago, April 6-8. Dr. Chester C. Guy, 5800 Stony Island Ave., Chicago 37, Secretary.

AMERICAN ASSOCIATION FOR THE STUDY OF NEOPLASTIC DISEASES, Lord Baltimore Hotel, Baltimore, April 29-30. Dr. Bruce H. Sisler, P. O. Box 268, Gatlinburg, Tenn., Executive Secretary.

AMERICAN ASSOCIATION FOR THORACIC SURGERY, Sheraton-Mt. Royal Hotel, Montreal, Canada, May 3-5. Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif., Secretary.

AMERICAN COLLEGE OF ALLERGISTS, Roney Plaza Hotel, Miami Beach, Fla., April 5-10. Dr. Fred W. Wittich, 423 LaSalle Medical Bldg., Minneapolis 2, Secretary.

AMERICAN COLLEGE OF PHYSICIANS, Conrad Hilton Hotel, Chicago, April 5-9. Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Executive Secretary.

AMERICAN DERMATOLOGICAL ASSOCIATION, The Greenbrier, White Sulphur Springs, W. Va., April 13-17. Dr. J. Lamar Callaway, Duke Hospital, Durham, N. C., Secretary.

AMERICAN FEDERATION FOR CLINICAL RESEARCH, Haddon Hall, Atlantic City, N. J., May 2. Dr. Lawrence E. Hinkle Jr., 525 East 68th St., New York 21, Secretary.

AMERICAN GOITER ASSOCIATION, The Somerset, Boston, April 29-May 1. Dr. John C. McClintock, 149½ Washington Ave., Albany 10, N. Y., Secretary.

AMERICAN HEART ASSOCIATION, Conrad Hilton Hotel, Chicago, April 1-4. Dr. William H. Bunn, 44 East 23d St., New York 10, Secretary.

AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, Hotel Statler, Boston, March 27. Dr. Louis J. Feit, 66 Park Ave., New York, Secretary.

AMERICAN PEDIATRIC SOCIETY, The Inn, Buck Hill Falls, Pa., May 3-5. Dr. A. C. McGuinness, 237 Medical Laboratories, University of Pennsylvania, Philadelphia 4, Secretary.

AMERICAN PHYSIOLOGICAL SOCIETY, Ambassador Hotel, Atlantic City, N. J., April 10-16. Dr. Milton O. Lee, 2101 Constitution Ave., Washington 25, D. C., Executive Secretary.

AMERICAN PSYCHIATRIC ASSOCIATION, St. Louis, May 3-7. Dr. R. Finley Gayle Jr., 6300 Three Chopt Road, Richmond 21, Va., Secretary.

AMERICAN PSYCHOSOMATIC SOCIETY, Jung Hotel, New Orleans, March 27-28. Dr. Theodore Lidz, 333 Cedar St., New Haven 11, Conn., Secretary.

AMERICAN SOCIETY OF BIOLOGICAL CHEMISTS, Atlantic City, N. J., April 12-16. Dr. Philip Handler, Duke University, Durham, N. C., Secretary.

AMERICAN SOCIETY FOR CLINICAL INVESTIGATION, Haddon Hall, Atlantic City, N. J., May 3. Dr. William M. M. Kirby, Univ. of Washington School of Medicine, Seattle 5, Secretary.

AMERICAN SOCIETY FOR EXPERIMENTAL PATHOLOGY, Atlantic City, N. J., April 12-16. Dr. Cyrus C. Erickson, 874 Union Ave., Memphis 3, Tenn., Secretary.

AMERICAN SOCIETY OF MAXILLOFACIAL SURGEONS, Washington, D. C., April 25-28. Dr. Casper M. Epsteen, 25 East Washington St., Chicago 2, Secretary.

AMERICAN SOCIETY FOR PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, Atlantic City, N. J., April 12-16. Dr. Carl C. Pfeiffer, 1853 West Polk St., Chicago 12, Secretary.

AMERICAN SOCIETY FOR THE STUDY OF STERILITY, Roosevelt Hotel, New Orleans, May 13-15. Dr. Herbert H. Thomas, 920 South 19th St., Birmingham, Ala., Secretary.

AMERICAN SURGICAL ASSOCIATION, Hotel Cleveland, Cleveland, April 28-30. Dr. R. Kennedy Gilchrist, 59 East Madison St., Chicago 3, Secretary.

ARIZONA MEDICAL ASSOCIATION, San Marcos Hotel, Chandler, April 25-28. Dr. Dermont W. Melick, 541 Security Bldg., Phoenix, Secretary.

ARKANSAS MEDICAL SOCIETY, Goldman Hotel, Fort Smith, April 19-21. Dr. J. J. Monfort, 215 Kelley Bldg., Fort Smith, Secretary.

ASSOCIATION OF AMERICAN PHYSICIANS, Chalfonte-Haddon Hall, Atlantic City, N. J., May 4-5. Dr. W. Barry Wood Jr., 600 S. Kingshighway Blvd., St. Louis 10, Secretary.

CALIFORNIA MEDICAL ASSOCIATION, Hotel Biltmore, Los Angeles, May 9-13. Mr. John Hunton, 450 Sutter St., San Francisco 8, Executive Secretary.

CONFERENCE ON MICROCIRCULATORY PHYSIOLOGY AND PATHOLOGY, University of Texas, Galveston, Texas, April 8-9. Dr. Edward H. Bloch, Western Reserve University School of Medicine, Dept. of Anatomy, Cleveland 6, Chairman.

CONNECTICUT STATE MEDICAL SOCIETY, Bulkeley High School, Hartford, April 27-29. Dr. Creighton Barker, 160 St. Ronan St., New Haven, Executive Secretary.

EASTERN SECTION, AMERICAN CONGRESS OF PHYSICAL MEDICINE, Newark, N. J., April 10. Dr. H. L. Rudolph, 400 North Fifth St., Reading, Pa., Secretary.

EASTERN STATES HEALTH EDUCATION CONFERENCE, New York Academy of Medicine, New York, April 29-30. Dr. Iago Galdston, 2 East 103d St., New York 19, Secretary.

EASTERN SURGICAL SOCIETY, Boston, March 26-27. Dr. J. William Hinton, 130 East 79th St., New York, Secretary.

FEDERATION OF AMERICAN SOCIETIES for EXPERIMENTAL BIOLOGY, Convention Hall, Atlantic City, N. J., April 12-16. Dr. M. O. Lee, 2101 Constitution Ave., Washington 25, D. C., Secretary.

FLORIDA MEDICAL ASSOCIATION, Hollywood Beach Hotel, Hollywood, April 25-28. Dr. Samuel M. Day, P. O. Box 1018, Jacksonville, Secretary.

GEORGIA, MEDICAL ASSOCIATION OF, Hotel Dempsey and Macon Auditorium, Macon, May 2-5. Dr. David H. Poer, 875 West Peachtree St. N.E., Atlanta, Secretary.

HAWAII MEDICAL ASSOCIATION, Maybel Smyth Bldg., Honolulu, May 13-16. Dr. Samuel L. Yee, 510 S. Beretania St., Honolulu 13, Secretary.

INDUSTRIAL HEALTH CONFERENCE, Hotel Sherman, Chicago, April 24-30. Dr. E. C. Holmblad, 28 East Jackson Blvd., Chicago 4, Managing Director.

INDUSTRIAL MEDICAL ASSOCIATION, Hotel Sherman, Chicago, April 27-30. Dr. Arthur K. Peterson, 28 East Jackson Blvd., Suite 1300, Chicago 4, Secretary.

IOWA STATE MEDICAL SOCIETY, Des Moines, April 25-28. Dr. Allan B. Phillips, 529 36th St., Des Moines 12, Secretary.

ISTHMIAN CANAL ZONE, MEDICAL ASSOCIATION OF THE, El Panama Hotel, Panama City, R. P., March 24-26. Dr. I. Robert Berger, Box "A," Balboa Heights, Canal Zone.

JOHN A. ANDREW CLINICAL SOCIETY, Memorial Hospital, Tuskegee Institute, Ala., April 11-16. Dr. Eugene H. Dibble Jr., John A. Andrew Memorial Hospital, Tuskegee Institute, Ala., Secretary.

KANSAS MEDICAL SOCIETY, Hotel Jayhawk, Topeka, May 2-6. Dr. D. D. Vermillion, 315 West Fourth St., Topeka, Secretary.

MARYLAND, MEDICAL AND CHIRURGICAL FACULTY OF THE STATE OF, Baltimore, April 27-28. Dr. Everett S. Diggs, 1211 Cathedral St., Baltimore, Secretary.

MISSOURI STATE MEDICAL ASSOCIATION, Hotel Jefferson, St. Louis, April 4-7. Dr. E. Royce Bohrer, 634 North Grand Blvd., St. Louis 3, Secretary.

NATIONAL CONFERENCE ON HEALTH IN COLLEGES, Hotel Statler, New York, May 5-8. Miss Charlotte V. Leach, 12th Floor, 1790 Broadway, New York 19, Secretary.

NEBRASKA STATE MEDICAL ASSOCIATION, Hotel Cornhusker, Lincoln, May 10-13. Dr. R. B. Adams, 1315 Sharp Bldg., Lincoln 8, Secretary.

NEW MEXICO MEDICAL SOCIETY, Hotel LaFonda, Santa Fe, May 13-15. Mr. R. R. Marshall, 221 West Central Ave., Albuquerque, Executive Secretary.

NEW YORK, MEDICAL SOCIETY OF THE STATE OF, Hotel Statler, New York, May 10-14. Dr. Walter P. Anderton, 386 Fourth Avenue, New York 16, Secretary.

NORTH CAROLINA, MEDICAL SOCIETY OF THE STATE OF, Hotel Carolina, Pinehurst, May 3-5. Dr. Millard D. Hill, 203 Capital Club Bldg., Raleigh, Secretary.

NORTH DAKOTA STATE MEDICAL ASSOCIATION, Grand Forks, May 1-4. Mr. Lyle A. Limond, Box 1198, Bismarck, Executive Secretary.

OHIO STATE MEDICAL ASSOCIATION, Columbus, April 13-15. Mr. Charles S. Nelson, 79 East State St., Columbus 15, Executive Secretary.

OKLAHOMA STATE MEDICAL ASSOCIATION, Municipal Auditorium, Oklahoma City, May 9-12. Mr. R. H. Graham, 1227 Classen Drive, Oklahoma City, Executive Secretary.

PACIFIC NORTHWEST SOCIETY OF PATHOLOGISTS, Amphitheater, Dept. of Pathology, University of Washington School of Medicine, Seattle, April 23-24. Dr. John L. Whitaker, 315 South K St., Tacoma 3, Wash., Secretary.

PACIFIC NORTHWEST SOCIETY OF PLASTIC AND RECONSTRUCTIVE SURGEONS, Seattle, April 3. Dr. E. E. Banfield, Medical Arts Bldg., Tacoma 2, Wash., Secretary.

RHODE ISLAND MEDICAL SOCIETY, Rhode Island Medical Society Library, Providence, May 4-5. Dr. Thomas Perry Jr., 106 Francis St., Providence 3, Secretary.

SECTIONAL MEETINGS, AMERICAN COLLEGE OF SURGEONS:
MONTREAL, CANADA, Mount Royal Hotel, March 31-April 2. Dr. Harry S. Morton, 900 Sherbrooke St., West, Montreal, Canada, Chairman.

SOCIETY OF AMERICAN BACTERIOLOGISTS, William Penn Hotel, Pittsburgh, May 2-7. Dr. John Hays Bailey, Sterling-Winthrop Research Institute, Rensselaer, N. Y., Secretary.

SOCIETY OF NEUROLOGICAL SURGEONS, The Waldorf Astoria, New York, April 23-24. Dr. Edgar F. Fincher, Emory University, Ga., Secretary.

SOCIETY FOR PEDIATRIC RESEARCH, Buck Hill Falls Inn, Buck Hill Falls, Pa., May 4-6. Dr. Sydney S. Gellis, 330 Brookline Ave., Boston 15, Secretary.

SOUTH CAROLINA MEDICAL ASSOCIATION, Ocean Forest Hotel, Myrtle Beach, May 11-13. Dr. Robert Wilson, 165 Rutledge Ave., Charleston, Secretary.

SOUTHEASTERN ALLERGY ASSOCIATION, Dinkler-Plaza Hotel, Atlanta, Ga., March 25-27. Dr. Katharine B. MacInnis, 1515 Bull St., Columbia 1, S. C., Secretary.

SOUTHWEST ALLERGY FORUM, Roosevelt Hotel, New Orleans, May 9-11. Dr. Stanley Cohen, S. W. Allergy Forum, 1441 Delachaise St., New Orleans 15, Secretary.

STUDENT AMERICAN MEDICAL ASSOCIATION, Sherman Hotel, Chicago, May 1-3. Mr. Russell F. Staudacher, 535 N. Dearborn St., Chicago 10, Executive Secretary.

TENNESSEE STATE MEDICAL ASSOCIATION, Nashville, April 18-21. Mr. V. O. Foster, 706 Church St., Nashville 3, Executive Secretary.

TEXAS MEDICAL ASSOCIATION, Gunter Hotel, San Antonio, May 3-5. Dr. J. M. Travis Sr., 1801 Lamar Blvd., Austin, Secretary.

UNITED STATES-MEXICO BORDER PUBLIC HEALTH ASSOCIATION, Albuquerque, N. Mex., April 6-9. Sidney B. Clark, 314 U. S. Court House, El Paso, Texas, Secretary.

WESTERN BRANCH, AMERICAN PUBLIC HEALTH ASSOCIATION, Olympic Hotel, Seattle, May 9-12. Mrs. L. Amy Darter, Division of Laboratories, State Dept. of Public Health, Berkeley, Calif., Secretary.

WESTERN INDUSTRIAL MEDICAL ASSOCIATION, Hotel Biltmore, Los Angeles, May 8. Dr. Edward J. Zaik, 740 S. Olive St., Los Angeles 14, Secretary.

FOREIGN AND INTERNATIONAL

ASSOCIATION OF SURGEONS OF GREAT BRITAIN AND IRELAND, Leeds, England, May 13-15, 1954. Dr. Henry W. S. Wright, 45 Lincoln's Inn Fields, London W.C.2, England, Honorary Secretary.

BRITISH MEDICAL ASSOCIATION, Glasgow, Scotland, July 1-9, 1954. Dr. A. Macrae, B.M.A. House, Tavistock Square, London, W.C.1, England, Secretary.

CANADIAN MEDICAL ASSOCIATION, Vancouver, B. C., Canada, June 14-18, 1954. Dr. T. C. Routley, 244 St. George St., Toronto 5. Ontario, Canada, General Secretary.

CONFERENCE OF INTERNATIONAL UNION AGAINST TUBERCULOSIS, Madrid, Spain, Sept. 26-Oct. 2, 1954. Secretariat, Escuela de Tisiologia, Ciudad Universitaria, Madrid, Spain.

CONGRESS OF INTERNATIONAL ASSOCIATION FOR THE PREVENTION OF BLINDNESS, New York, N. Y., U. S. A., Sept. 12-17, 1954. Professor Franceschetti, 2 Avenue Mirmot, Geneva, Switzerland, Secretary-General.

CONGRESS OF INTERNATIONAL ASSOCIATION FOR THE STUDY OF THE BRONCHI, Geneva, Switzerland, June 5-6, 1954. Professor A. Montandon, Clinique Universitarie d O.R.L., Hôpital Cantonal, Geneva, Switzerland, Chairman.

EUROPEAN SOCIETY OF CARDIOVASCULAR SURGERY, Edinburgh, Scotland, July 9-10, 1954. For information address: Mr. A. J. Slessor, Department of Surgery, University New Building, Edinburgh 8, Scotland.

HEALTH CONGRESS OF THE ROYAL SANITARY INSTITUTE, Scarborough, England, April 27-30, 1954. Mr. P. Arthur Wells, Royal Sanitary Institute, 90 Buckingham Palace Road, London, S.W.1, England, Secretary.

INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, Los Angeles, Calif., U. S. A., Oct. 10-14, 1954. For information write: Dr. T. H. Seldon, 102-110 Second Avenue S.W., Rochester, Minn., U. S. A.

INTERNATIONAL CANCER CONGRESS, Sao Paulo, Brazil, July 23-29, 1954. Prof. A. Prudente, 171 rua Benjamin Constante, Sao Paulo, Brazil, President.

INTERNATIONAL CONFERENCE ON THROMBOSIS AND EMBOLISM, Basle, Switzerland, July 20-24, 1954. Dr. W. Merz, Chief Medical Officer, Gynecological Clinic, University of Basle, Basle, Switzerland, Hon. Secretary.

INTERNATIONAL CONGRESS OF CLINICAL PATHOLOGY, Washington, D. C., U. S. A., Sept. 6-10, 1954. Dr. Robert A. Moore, Washington University School of Medicine, St. Louis 10, Mo., U. S. A., Chairman, Committee on Arrangements.

INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST, Barcelona, Spain, Oct. 4-8, 1954 Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Ill., U. S. A., Executive Secretary.

INTERNATIONAL CONGRESS ON GROUP PSYCHOTHERAPY, Toronto, Ont., Canada, Aug. 12-14, 1954. Dr. J. L. Moreno, Room 327, 101 Park Ave., New York 17, N. Y., U. S. A., Director of Organizing Committee.

INTERNATIONAL CONGRESS ON GYNECOLOGY AND OBSTETRICS, Geneva, Switzerland, July 26-31, 1954. Dr. H. de Watteville, Maternité Hôpital Cantonal, Geneva, Switzerland, President.

INTERNATIONAL CONGRESS OF HEMATOLOGY, Paris, Sept. 6-11, 1954. Dr. Jean Bernard, 86 rue d'Assas, Paris 6e, France, Secretary.

INTERNATIONAL CONGRESS OF THE HISTORY OF MEDICINE, Rome and Salerno, Italy, Sept. 13-20, 1954. For information write: Segreteria XIV Congresso Internazionale di Storia della Medicina, Instituto di Storia della Medicine, Citta Universitaria, Rome, Italy.

INTERNATIONAL CONGRESS OF HYDROCLIMATISM AND THALASSOTHERAPY, Dubrovnik, Yugoslavia, May 8-16, 1954. Prof. C. Plavsic, Zeleni venac 1, Belgrade, Yugoslavia, Secretary General.

INTERNATIONAL CONGRESS OF INDUSTRIAL MEDICINE, Naples, Italy, Sept. 13-19, 1954. Professor Scipione Caccuri, Director, Institute of Industrial Medicine Policlinico, Naples, Italy, Chairman, Organizing Committee.

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE, Stockholm, Sweden, Sept. 15-18, 1954. Professor Anders Kristenson, Karolinska Sjukhuset, Stockholm 60, Sweden, Secretary-General.

INTERNATIONAL CONGRESS OF INTERNATIONAL COLLEGE OF SURGEONS, São Paulo, Brazil, April 26-May 2, 1954. Dr. Max Thorek, 1516 Lake Shore Drive, Chicago, Ill., U. S. A., Secretary-General.

INTERNATIONAL CONGRESS ON MENTAL HEALTH, University of Toronto, Toronto, Ontario, Canada, Aug. 14-21, 1954. For information write: Executive Officer, International Congress on Mental Health, 111 St. George St., Toronto, Ontario, Canada.

INTERNATIONAL CONGRESS OF MILITARY MEDICINE AND PHARMACY, Buenos Aires, Argentine, April 21-28, 1954. Direcion General de Sanidad Militar, Pozos 2045, Buenos Aires, Argentine.

INTERNATIONAL CONGRESS OF NUTRITION, Amsterdam, Netherlands, Sept. 13-17, 1954. Dr. M. van Eekelen, Centraal Instituut voor Voedingsonderzoek T.N.O., 61 Catharynesingel, Utrecht, Netherlands, General Secretary.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, University of Montreal and McGill University, Montreal, Canada, Sept. 9-11, 1954, and Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 12-17, 1954. Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn., U. S. A., Secretary-General.

INTERNATIONAL CONGRESS OF ORTHOPEDIC SURGERY AND TRAUMATOLOGY, Berne, Switzerland, Aug. 30-Sept. 3, 1954. For information write: Professor M. Dubois, Isle-Hospital, Berne, Switzerland.

INTERNATIONAL CONGRESS OF PSYCHOLOGY, Montreal, Canada, June 7-12, 1954. For information write: Prof. H. S. Langfeld, International Union of Scientific Psychology, Eno Hall, Princeton University, Princeton, N. J., U. S. A.

INTERNATIONAL CONGRESS FOR PSYCHOTHERAPY, Zurich, Switzerland, July 21-24, 1954. Dr. H. K. Fierz, Theaterstrasse 12, Zurich 1, Switzerland, Secretary General.

INTERNATIONAL CONGRESS OF THE SOCIEDAD DE MÉDICOS INTERNOS, Residentes y Becarios del Instituto Nacional de Cardiologia de Mexico, Acapulco, Mexico, April 21-24, 1954. For information address: Dr. Jorge Soberón Acevedo, Avenida, Cuauhtemoc No. 300, Mexico, D. F., Mexico.

INTERNATIONAL GERONTOLOGICAL CONGRESS, London and Oxford, England, July 12-22, 1954. Prof. R. E. Tunbridge, General Infirmary, Department of Medicine, The University, Leeds, England, President.

INTERNATIONAL INSTITUTE ON CHILD PSYCHIATRY, Toronto, Canada, Aug. 13-14, 1954. Miss Helen Speyer, International Association for Child Psychiatry, 1790 Broadway, New York 19, N. Y., U. S. A., Executive Officer.

INTERNATIONAL POLIOMYELITIS CONGRESS, Rome, Italy, Sept. 6-10, 1954. Mr. Stanley E. Henwood, 120 Broadway, New York 5, N. Y., U. S. A., Executive Secretary.

INTERNATIONAL SOCIETY OF ANGIOLOGY, North American Chapter, Hotel Mark Hopkins, San Francisco, Calif., U. S. A., June 19, 1954. Dr. Henry Haimovici, 105 East 90th St., New York, N. Y., U. S. A., Secretary.

INTERNATIONAL SOCIETY OF BLOOD TRANSFUSION, Paris, France, Sept. 12-19, 1954. For information write: Colonel Julliard, Société Internationale de Transfusion Sanguine, 53 Boulevard Diderot, Paris 12°, France.

INTERNATIONAL SOCIETY FOR CELL BIOLOGY, Leiden, Netherlands, Sept. 2-9, 1954. Professor Peter J. Gaillard, University of Leiden, Leiden, Netherlands, Secretary.

INTERNATIONAL SOCIETY OF GEOGRAPHICAL PATHOLOGY, Washington, D. C., U. S. A., Sept. 6-10, 1954. Professor Fred C. Roulet, Hebelstrasse 24, Basle, Switzerland, Secretary-General.

IRISH MEDICAL ASSOCIATION, Killarney, Ireland, July 7-10, 1954. Dr. P. J. Delaney, 10, Fitzwilliam Place, Dublin, Ireland, Medical Secretary.

JOURNEES MEDICALES, Paris, France, April 21-25, 1954. For information write: Secretariat of the Journees, 12, rue Pierre-Geofroix, Colombes (Seine) France.

LATIN AMERICAN CONGRESS ON GYNECOLOGY AND OBSTETRICS, Sao Paulo, Brazil, July 10-15, 1954. Prof. Dr. Jairo Ramos, av. Brigaderio Luiz Antonio, 278-8° andar, Sao Paulo, Brazil, Chairman of Organizing Committee of Medical Congresses.

LATIN AMERICAN CONGRESS ON MENTAL HEALTH, Sao Paulo, Brazil, July 17-22. For information address: Professor A. C. Pacheco e Silva, Avenida Brigadeiro Luiz Antonio 651, Sao Paulo, Brazil.

MEDICAL WOMEN'S INTERNATIONAL ASSOCIATION CONGRESS, Lake Garda, Italy, Sept. 15-21, 1954. Dr. Ada Chree Reid, 118 Riverside Drive, New York 24, N. Y., U. S. A., President.

PAN AMERICAN CONGRESS OF CHILD WELFARE AND PEDIATRICS, Sao Paulo, Brazil, July 15-21, 1954. For information address: Dr. Jairo Ramos, Avenida Brigaderio Luiz Antonio 278-8° andar, Sao Paulo, Brazil.

PAN AMERICAN CONGRESS OF GASTROENTEROLOGY, Sao Paulo, Brazil, July 19-24, 1954. For information address: Dr. Jairo Ramos, Avenida Brigadeiro Luiz Antonio 278-8° andar, Sao Paulo, Brazil.

PAN AMERICAN CONGRESS OF OPHTHALMOLOGY (Interim), São Paulo, Brazil, June 17-21, 1954. Dr. Moacyr E. Alvaro, Consolacao 1151, São Paulo, Brazil, President.

PAN AMERICAN CONGRESS OF VETERINARY MEDICINE, Sao Paulo, Brazil, April 3-10, 1954. Dr. Joao Soares Veiga, Rua Pires da Mota 159, Sao Paulo, Brazil, Chairman of Organizing Committee

PAN-PACIFIC SURGICAL CONGRESS, Honolulu, Hawaii, Oct. 7-18, 1954. Dr. F. J. Pinkerton, Suite 7, Young Bldg., Honolulu 13, Hawaii, Director General.

SECTIONAL MEETING, AMERICAN COLLEGE OF SURGEONS, London, England, May 17-19, 1954. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Ill., U. S. A., Secretary.

SOUTH AMERICAN CONGRESS OF ANGIOLOGY, Sao Paulo, Brazil, July 1954. For information write: Dr. Rubens Carlos Mayall, Rua Senador Vergueiro 73, Rio de Janeiro, Brazil, S. A.

WORLD CONGRESS OF CARDIOLOGY, Washington, D. C., and Bethesda, Md., U. S. A., Sept. 12-17, 1954. Dr. L. W. Gorham, 44 East 23d St., New York 10, N. Y., U. S. A., Secretary-General.

WORLD CONGRESS OF INTERNATIONAL SOCIETY FOR THE WELFARE OF CRIPPLES, Scheveningen-The Hague, Netherlands, Sept. 13-17, 1954. Secretariat: Miss H. P. Post, Pieter Lastmarkade 37, Amsterdam Z, Netherlands.

WORLD FEDERATION OF OCCUPATIONAL THERAPISTS, Edinburgh, Scotland, August 17, 1954.

WORLD MEDICAL ASSOCIATION, Rome, Italy, Sept. 26-Oct. 2, 1954. Dr. Louis H. Bauer, 345 East 46th St., New York 17, N. Y., U. S. A., Secretary-General.

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS: *Parts I and II.* Held in approved medical schools where there are five or more candidates. Dates: Feb. 9-10; April 20-21 (Part II only); June 22-23; Sept. 7-8 (Part I only). Candidates may file examinations at any time but the National Board must receive them at least six weeks before the date of the examination they wish to take. New candidates should apply by formal registration; registered candidates should notify the board by letter. Sec., Dr. John P. Hubbard, 133 S. 36th St., Philadelphia 4.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 22-24, 1954. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery.

ARKANSAS:* *Examination.* Little Rock, June 10-11. Sec., Dr. Joe Verser, Harrisburg. *Eclectic.* Little Rock, June 11-12. Sec., Dr. O. L. Atkinson, 2528 Central Ave., Hot Springs National Park.

CALIFORNIA: *Written.* Los Angeles, March 15-18; San Francisco, June 21-24; San Diego, Aug. 23-26; Sacramento, Oct. 18-21. *Oral.* Los Angeles, March 13; San Francisco, June 19; San Diego, Aug. 21; Los Angeles, Nov. 20. *Oral and Clinical Examination for Foreign Medical School Graduates.* Sec., Dr. Louis E. Jones, 1020 N Street, Sacramento.

COLORADO:* *Examination.* Denver, June 8-9. Final date for filing applications is May 8. *Reciprocity.* Denver, April 13. Final date for filing applications is March 13. Exec. Sec., Mrs. Beulah H. Hudgens, 831 Republic Bldg., Denver 2.

DISTRICT OF COLUMBIA:* *Examination.* Washington, May 10-11. Deputy Director, Mr. Paul Foley, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington, D. C.

FLORIDA:* *Examination.* Jacksonville, June 27-29. Sec., Dr. Homer L. Pearson, 901 N.W. 17th St., Miami.

GEORGIA: *Examination and Reciprocity.* Atlanta and Augusta, June. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

GUAM: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Director of Medical Services, Guam Memorial Hospital, Agana

ILLINOIS: *Examination and Reciprocity.* Chicago, April 6-8; June 22-24, and Oct. 5-7. Supt. of Registration, Mr. Fredric B. Selcke, Capitol Bldg., Springfield.

INDIANA: *Examination.* Indianapolis, June 16-18. Exec. Sec., Miss Ruth V. Kirk, 538 K of P Bldg., Indianapolis.

IOWA:* *Examination.* Iowa City, June 14-16. Sec., Dr. M. A. Royal, 506 Fleming Bldg., Des Moines.

KANSAS: *Examination and Reciprocity.* Kansas City, June 9-10. Sec., Dr. O. W. Davidson, 872 New Brotherhood Bldg., Kansas City.

KENTUCKY: *Examination.* Louisville, June 7-9. Address Mr. Raymond F. Nixon, Assistant Secretary, 620 S. 3rd St., Louisville 2.

MARYLAND: *Examination.* Baltimore, June 15-18. Sec., Dr. E. H. Kloman, 1215 Cathedral St., Baltimore 1.

MICHIGAN:* *Examination.* Detroit and Ann Arbor, June 1954. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing 8.

MISSISSIPPI: *Examination and Reciprocity.* Jackson, June. Asst. Sec., Dr. R. N. Whitfield, Old Capitol, Jackson 113.

MONTANA: *Reciprocity.* Helena, April 5. *Examination.* Helena, April 6-7. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

NEBRASKA:* *Examination.* Omaha, June 1954. Director, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

NEW JERSEY: *Examination,* Trenton, June 15-18. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO:* *Examination and Reciprocity.* Santa Fe, April 12-13. Sec., Dr. R. C. Derbyshire, 227 E. Palace Ave., Santa Fe.

OHIO: *Examination.* Columbus, June 14-16. *Reciprocity.* Columbus, April 13. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA:* *Examination.* Oklahoma City, June 9-10. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

OREGON:* *Examination.* Portland, April 23. Final date for filing applications is March 22. Ex. Sec., Mr. Howard I. Bobbitt, 609 Failing Bldg., Portland.

SOUTH DAKOTA:* Reciprocity can be obtained at any time between meetings of board. Executive Secretary, Mr. John C. Foster, 300 First National Bank Bldg., Sioux Falls.

TENNESSEE:* *Examination.* Memphis, March 24-25. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

TEXAS: *Examination and Reciprocity.* Fort Worth, June 21-23. Rec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Ft. Worth 2.

UTAH: *Examination.* Salt Lake City, July 7-9. Final date for filing application is June 15. Director, Mr. Frank E. Lees, Department of Business Regulation, 314 State Capitol, Salt Lake City.

VIRGINIA: *Examination and Reciprocity.* Richmond, June 16. Address: Virginia Board of Medical Examiners, 631 First St., S.W., Roanoke.

ALASKA:* On application. Sec., Dr. W. M. Whitehead, 172 South Franklin St., Juneau.

VIRGIN ISLANDS: *Examination.* St. Thomas, June 9-10. Sec., Dr. Earle M. Rice, St. Thomas.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ALASKA: On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

ARKANSAS: *Examination.* Little Rock, May 3-4. Sec., Mr. Louis E. Gebauer, 1002 Donaghey Bldg., Little Rock.

FLORIDA: *Examination.* Gainesville, June. Sec., Mr. M. W. Emmel, University of Florida, Box 340, Gainesville.

NEBRASKA: *Examination.* Omaha, May 4-5. Director, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln 9.

OKLAHOMA: *Examination.* Oklahoma City, April 16-17. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

OREGON: *Examination.* Portlannd, June 5, Sept. 11 and Dec. 4. Sec., Mr. Charles D. Byrne, State Board of Higher Education, Eugene.

SOUTH DAKOTA: *Examination.* June 11-12. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.

TEXAS: *Examination.* April 1954. Location determined by number of applicants from different sections of the state. Sec., Brother Raphael Wilson, C.S.C., 407 Perry-Brooke Bldg., Austin.

WISCONSIN: *Examination.* Madison, April 3 and June 5. Final date for filing application is March 26 and May 28. Sec., Dr. W. H. Barber, 621 Ransom St., Ripon.

*Basic Science Certificate required.

DEATHS

Herrick, James Bryan ⊕ Chicago, a recipient of the Distinguished Service Medal of the American Medical Association, died in the Presbyterian Hospital March 7, aged 92, of carcinoma of the stomach and peritonitis. Dr. Herrick was born in Oak Park, Ill., Aug. 11, 1861. He attended Oak Park High School and the Rock River Seminary at Mount Morris, Ill., and was graduated from the University of Michigan, Ann Arbor, in 1882. For a time he taught in the Central High School, Peoria, Ill., acquiring there a love of good literature and a facility for expressing himself in the English language. Through his association with Moses Coit Tyler he developed a great interest in Chaucer. Dr. Herrick then took up the study of medicine, receiving the degree of doctor of medicine from Rush Medical College in 1888. On completing his internship at Cook County Hospital, he joined the staff of his alma mater, serving as instructor in medicine from 1890 to 1894, adjunct professor from 1894 to 1900, professor from 1900 to 1927 and then became emeritus. In 1930 a fellowship was named in his honor at Rush Medical College by Dr. Frank Billings. He had been a member of the staff of Presbyterian Hospital since 1890, serving as president of the medical board from 1908 to 1913 and as vice-president from 1903 to 1907. He was attending physician to the Presbyterian Hospital from 1890 to 1924 when he was made consultant. A bas-relief portrait of him was presented to the hospital Nov. 6, 1943, as a feature of special anniversary exercises. The first electrocardiograph used in Chicago was in his office, and he was instrumental in having installed in the Presbyterian Hospital the first electrocardiograph used in any hospital in the city. He was identified with the Central Free Dispensary for many years and for a time was chairman of the medical advisory committee of Sunset Camp, a convalescent colony sponsored by the Chicago Heart Association. Herrick House, a camp for rheumatic heart victims in Fox River Valley, was named for him. A member of many scientific societies and a specialist certified by the American Board of Internal Medicine, Dr. Herrick served as president of the Society of Medical History of Chicago, American Association for the History of Medicine, Association of American Physicians, American Heart Association, which in 1951 presented him with a gold heart award, and of the Congress of American Physicians and Surgeons. In 1939, after concluding a year as vice-president of the American College of Physicians, the college conferred on him the title "master" for the first time in 11 years that the honor had been accorded. He was a founder of the Institute of Medicine of Chicago and active on its board of governors since 1915, holding the presidency in 1925. In October, 1949, several hundred fellows of the institute and prominent guests paid tribute to Dr. and Mrs. Herrick at a reception to mark the publication of Dr. Herrick's autobiography "Memories of Eighty Years." He was a member of the board of trustees of the Lewis Institute and the McCormick Memorial Institute, a past president of the Chicago Pathological Society and the Chicago Literary Club, and founder and first president of the Chicago Heart Association. He was founder and first president of the Chicago Society of Internal Medicine, and a member of the Central Society for Research. In 1935 a paper was prepared by Dr. William H. Holmes for the Central Interurban Clinical Club of which Dr. Herrick was an honorary member, reviewing his distinguished career as a student, teacher, and scientist. Notable awards were presented to Dr. Herrick, including honorary memberships and degrees from institutions and societies. In 1930 he received the Kober medal of the Association of American Physicians for research in scientific medicine, and in 1933 the New York Academy of Medicine elected him to honorary fellowship. He also received the "Gold Headed Cane" from the University of California Medical School in San Francisco for his accomplishments in the practice of medicine, in teaching, and in investigation. He gave such prominent lectures as the Fenger, George W. Gay, Harvey, and Abner Wellborn Calhoun lectures. In 1939, the American Medical Association, of which he was an Associate Fellow and which he served as a member of the Judicial Council from 1928 to 1934, presented its Distinguished Service Medal to Dr. Herrick for his work on coronary thrombosis. A renowned internist, his articles on this subject are said to have done more to force clinical recognition of the conditions and to stimulate clinical and experimental study than all other writings on the subject. Probably most significant was the contribution "Clinical Features of Sudden Obstruction of the Coronary Arteries," published in THE JOURNAL, Dec. 7, 1912. He first described sickle cell anemia as a clinical entity in 1910. He was the author of "Handbook of Medical Diagnosis" and "A Short History of Cardiology." In 1907 he received the honorary degree of master of arts from the University of Michigan, which in 1932 awarded him the honorary degree of doctor of laws. In January, 1939, at the 194th convocation of the University of Chicago, the honorary doctorate of science was conferred on him and in June, 1940, the honorary degree of doctor of science was awarded to him by Northwestern University.

Burrier, Walter Painter ⊕ Brookline, Mass.; born in Biddeford, Maine, April 12, 1882; Tufts College Medical School, Boston, 1908, specialist certified by the American Board of Psychiatry and Neurology; commissioned a surgeon in the U. S. Public Health Service from 1921 to 1924 when he joined the U. S. Veterans Bureau; lieutenant colonel in the Army of the United States from 1944 to 1946; formerly clinical director and executive officer at the Bronx Hospital in New York, where he was chief of the outpatient department; clinical director and executive officer, West Roxbury (Mass.) Hospital from 1924 to 1928; clinical director, Bedford (Mass.) Hospital from 1928 to 1934; chief of the neuropsychiatric division, Diagnostic Center, Washington, D. C., 1934-1935; clinical director at Veterans Administration Hospital, Northampton, Mass., from 1935 to 1937; clinical director from 1937 to 1945 and manager from 1945 to 1947 at the Veterans Administration Hospital in Bedford, Mass.; member of the Association of Military Surgeons of the United States, New England Society of Psychiatry, and the American Psychiatric Association; died in the Veterans Administration Hospital in West Roxbury, Jan. 28, aged 71, of carcinoma of the prostate.

Meyers, Milton Kayton ⊕ Philadelphia; born in Philadelphia, Jan. 3, 1882; University of Pennsylvania Department of Medicine, Philadelphia, 1902; specialist certified by the American Board of Psychiatry and Neurology; instructor in child psychiatry at Jefferson Medical College; member of the Philadelphia Neurological Society, Pathological Society of Philadelphia, Northern Medical Society of Philadelphia, Anthropological Society, and Philadelphia Psychiatric Society; attending psychatrist, Philadelphia Psychiatric Hospital; consulting neurologist, Jewish Hospital; neurologist, Northern Liberties Hospital; neuropsychiatrist, American Oncologic Hospital, assistant neurologist, Philadelphia General Hospital; translator and editor of Falta's "Endocrine Diseases, Including Their Treatment"; editor of "Lang's German-English Medical Dictionary"; contributor of the chapter on nervous diseases in Loewenberg's "Diagnostic Methods and Interpretations in Internal Medicine"; died Jan. 25, aged 72, of a heart attack.

Guequierre, Jacques Pierre ⊕ Bryn Mawr, Pa.; born in Milwaukee Nov. 6, 1891; Marquette University School of Medicine, Milwaukee, 1924; specialist certified by the American Board of Dermatology and Syphilology; member of the American Academy of Dermatology and Syphilology; associate professor of dermatology and syphilology at Temple University School of Medicine and the Medico-Chirurgical College, Graduate School of Medicine, University of Pennsylvania, Philadelphia; member and past president of the Philadelphia Dermatological Society; corresponding member of the Cuban Dermatological Society and the Cuban Cancer Society; chief of clinic, department of dermatology and syphilology at Temple University Hospital; chief of clinic in the department of physical therapy at Graduate

⊕ Indicates Member of the American Medical Association.

Hospital; consultant of dermatology at the Philadelphia State Hospital; affiliated with Bryn Mawr Hospital, where he died Jan. 15, aged 62, of coronary thrombosis and pneumonia.

Blydenburgh, George Theron ⊕ Delaware, Ohio; born in Brooklyn April 16, 1891; Cornell University Medical College, New York, 1918; assistant surgeon in the U. S. Naval Reserves at the Brooklyn Naval Hospital, 1918-1919; resident physician, Red Cross Hospital, New York, 1919-1920; member of the American Public Health Association; formerly secretary-treasurer of the American Student Health Association; from 1945 to 1952 on the board of directors of the Ohio Tuberculosis and Health Association; at one time a medical missionary, serving as superintendent of the Susan Toy Ensign Memorial Hospital in Nanchang, China from 1920 to 1931; assistant physician, New York State Department of Mental Hygiene, 1932-1933; Ohio Wesleyan University physician and director of student health service since 1933; died Jan. 22, aged 62, of congestive heart failure.

Kirkpatrick, William David ⊕ Bellingham, Wash.; born in Chelsea, Mass., March 10, 1872; University of Minnesota College of Homeopathic Medicine and Surgery, Minneapolis, 1895; member of the Pacific Coast Surgical Association, of which he was one of the founders; fellow of the American College of Surgeons; past president of the Washington State Medical Association; medical officer both for the American Red Cross and the U. S. Army during World War I; cited by Queen Marie of Roumania for meritorious services; for many years trustee for the Western Washington College of Education; affiliated with St. Luke's Hospital and St. Joseph's Hospital, where he died Jan. 13, aged 81, of coronary occlusion.

Arkebauer, Charles A. ⊕ Van Buren, Ark.; Missouri Medical College, St. Louis, 1899; formerly on the faculty of the University of Arkansas School of Medicine in Little Rock; member of the American Psychiatric Association; for many years affiliated with the State Hospital for Nervous Diseases in Little Rock; died in the Crawford County Hospital Oct. 22, aged 86.

Artis, G. Hubert, Los Angeles; Rush Medical College, Chicago, 1927; at one time practiced in Cedar Rapids, Iowa, where he was on the staffs of Mercy and St. Luke's Methodist hospitals; retired as medical examiner for the Baltimore and Ohio Railway in Cumberland, Md.; died in Centinela Valley Hospital in Inglewood, Calif., Dec. 12, aged 63, of cerebral hemorrhage.

Ayers, Thomas Wilburn, Atlanta, Ga.; College of Physicians and Surgeons, Baltimore, 1886; for many years a medical missionary in China; died Jan. 5, aged 95, of arteriosclerotic heart disease.

Barnard, Frank Steele, Los Angeles; Hahnemann Medical College and Hospital of Philadelphia, 1894; died Jan. 16, aged 94, of bronchopneumonia and arteriosclerosis.

Beers, Merritt Isaac ⊕ Middletown, N. Y.; New York Homeopathic Medical College and Hospital, New York, 1901; an Associate Fellow of the American Medical Association; on the staff of the Elizabeth A. Horton Memorial Hospital; died Dec. 11, aged 76, of arteriosclerotic heart disease.

Bender, Max, Union City, N. J.; Jefferson Medical College of Philadelphia, 1915; served on the staffs of the Christ Hospital in Jersey City and the North Hudson Hospital in Weehawken; died Jan. 11, aged 66, of coronary thrombosis.

Bickel, James T. ⊕ Lamar, Mo.; University Medical College of Kansas City, Mo., 1901; died Dec. 7, aged 81, of arteriosclerotic heart disease.

Biggers, John Alvin, Manhattan Beach, Calif.; University of Louisville (Ky.) Medical Department, 1907; veteran of the Spanish-American War; served on the staffs of the Los Angeles County General and Hollywood Presbyterian hospitals; died Jan. 7, aged 78, of lymphosarcoma.

Booth, William Telford ⊕ Coronado, Calif.; Emory University School of Medicine, 1930; resigned from the regular Navy Jan. 4, 1938; killed in an airplane crash Jan. 23, aged 48.

Bright, John Fulmer, Richmond, Va.; Medical College of Virginia, Richmond, 1898; emeritus professor of anatomy at his alma mater; served during World War I; mayor of Richmond from 1924 to 1940; died in the Johnston-Willis Hospital Dec. 29, aged 76, of aortic stenosis.

Brooks, Isaac William, Torrington, Wyo.; College of Physicians and Surgeons, Dallas, Texas, 1905; died Jan. 20, aged 80, of cerebral hemorrhage.

Burns, William Milton ⊕ North Little Rock, Ark.; University of Arkansas School of Medicine, Little Rock, 1914; for many years member of the school board; served as mayor; died Dec. 29, aged 75, of acute coronary thrombosis.

Bursheim, Peder Joseph ⊕ Des Moines, Iowa; Keokuk (Iowa) Medical College, College of Physicians and Surgeons, 1905; died in the Iowa Lutheran Hospital Jan. 17, aged 70, of carcinoma of the colon.

Butt, Arthur Parker Jr. ⊕ Elkins, W. Va.; University of Virginia Department of Medicine, Charlottesville, 1929; chief surgeon at Davis Memorial Hospital; died in Orlando, Fla., Dec. 16, aged 51, of coronary thrombosis.

Cafferky, Allan Bryan, Clarkston, Wash.; College of Medical Evangelists, Loma Linda and Los Angeles, 1945; served as a medical missionary; died in White Memorial Hospital in Los Angeles, Dec. 31, aged 33, of adenocarcinoma of the ascending colon.

Cardenas, Valentin Oriol ⊕ Orlando, Fla.; Universidad de la Habana Facultad de Medicina y Farmacia, Cuba, 1927; member of the Illinois State Medical Society; formerly practiced in Chicago, where he was affiliated with the Columbus Hospital; affiliated with the Orange Memorial Hospital; served during World War II; died Jan. 31, aged 50, in a plane crash.

Coffey, George W., Concordia, Kan.; Kansas City (Mo.) Medical College, 1893; died Jan. 19, aged 87, as the result of a fall.

Cooper, Benjamin F. ⊕ Detroit; Detroit College of Medicine, 1930; served during World War II; fellow of the American College of Surgeons; affiliated with Holy Cross Hospital and the East Side General Hospital; died in the Saratoga General Hospital Jan. 19, aged 47, of coronary occlusion.

Cozzolino, James Anthony, Hamden, Conn.; Jefferson Medical College of Philadelphia, 1920; on the staff of St. Raphael's Hospital in New Haven; died Jan. 12, aged 59, of myocardial infarction.

Danciger, Jacob Adolph ⊕ Memphis, Tenn.; University of Tennessee College of Medicine, Memphis, 1937; assistant professor of pediatrics at his alma mater; specialist certified by the American Board of Pediatrics; member of the American Pediatric Society; past president of the Memphis Pediatrics Society; served during World War II; died in John Gaston Hospital Nov. 24, aged 39, of chronic glomerulonephritis.

Day, George Huff ⊕ New Albany, Ind.; University of Louisville (Ky.) Medical Department, 1913; served in France during World War I; on the staff of St. Edward's Hospital; died Jan. 20, aged 63.

DeCanio, John ⊕ Talihina, Okla.; Marquette University School of Medicine, Milwaukee, 1940; member of the Medical Association of Montana; associated with the Indian Service; served during World War II; formerly head physician at the Claremore Indian Hospital in Claremore; medical officer in charge of Talihina Medical Center, U. S. Indian Hospital; died Dec. 3, aged 43, of multiple myeloma.

Dever, Neal, McAdoo, Pa.; Temple University School of Medicine, Philadelphia, 1911; also a lawyer; medical inspector for the McAdoo Borough School District for many years; died Dec. 13, aged 81, of senility.

Dezell, Earl R., Glendale, Calif.; Minneapolis College of Physicians and Surgeons, medical department of Hamline University, 1908; died Dec. 30, aged 70, of coronary occlusion and arteriosclerosis.

Dillman, Theodore Albert, Ann Arbor, Mich.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1908; died in Detroit Jan. 6, aged 77, of arteriosclerosis.

Frey, Albert ⊕ Buffalo; University of Buffalo School of Medicine, 1902; an Associate Fellow of the American Medical Association; affiliated with Deaconess Hospital; died Jan. 8, aged 74.

Gadol, William Norman ⊕ Bethesda, Md.; Syracuse University College of Medicine, 1948; formerly assistant surgeon in the U. S. Public Health Service Reserve; died Jan. 17, aged 35, of coronary occlusion.

Hayward, Herbert ⊕ Hamilton, Mont.; Milwaukee Medical College, 1908; fellow of the American College of Surgeons; served during World War I; chief surgeon, Marcus Daly Memorial Hospital, until his retirement two years ago; special consultant at the Rocky Mountain Laboratory; died suddenly Jan. 4, aged 71, of coronary thrombosis.

Hebble, Robert C., Springfield, Ohio; Starling Medical College, Columbus, 1895; past president of the Clark County Medical Society; on the courtesy staffs of the City Hospital and Mercy Hospital, where he died Dec. 5, aged 79, of coronary thrombosis.

Hochfeld, Herbert David ⊕ Seattle; Universität Bern Medizinische Fakultät, Switzerland, 1935; on the staff of the Virginia Mason Hospital, where he died Dec. 24, aged 45, of a postoperative infection.

Hoeffel, Adelaide Doolittle ⊕ Chicago; the Hahnemann Medical College and Hospital, Chicago, 1903; formerly on the faculty of her alma mater; on the courtesy staff of the Chicago Memorial Hospital, where she died Jan. 20, aged 86, of ruptured diverticulitis of the sigmoid colon, empyema of the gallbladder, and arteriosclerosis.

Hollingworth, Carl J., Portland, Ore.; University of Oregon Medical School, Portland, 1926; assistant clinical professor of medicine at his alma mater; a member of the board of directors of the Willamette University in Salem; formerly associated with the U. S. Public Health Service; died Jan. 14, aged 64, of rheumatic heart disease, coronary arteriosclerosis, and nephrosclerosis.

Horwitz, Sandor ⊕ Peoria, Ill.: Missouri Medical College, St. Louis, 1895; an Associate Fellow of the American Medical Association; member of the American Public Health Association; formerly district health officer for the state board of health, and health commissioner of Peoria; died in Chicago Jan. 17, aged 86, of coronary thrombosis.

Humphrey, Lester Hayden ⊕ Silver Springs, N. Y.; Albany (N. Y.) Medical College, 1900; served as health officer for Gainesville; for many years county coroner; member of the Wyoming County Welfare Department; on the staff of the Wyoming County Community Hospital in Warsaw, where he died Nov. 14, aged 82, of cerebral hemorrhage.

Inge, Francis Marion ⊕ Mobile, Ala.; Maryland Medical College, Baltimore, 1910; served during World War I; died in the Methodist Hospital, Houston, Texas, Jan. 13, aged 69, of abdominal aneurysm.

Lewin, Adolph Leo ⊕ Pittsburgh; Western Pennsylvania Medical College, Pittsburgh, 1892; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Germany, 1895; for many years member of the school board; affiliated with Western Pennsylvania, St. Francis, and Montefiore hospitals; died Dec. 31, aged 82, of coronary occlusion.

Lougee, William Wheeler ⊕ Malden, Mass.; New York University Medical College, New York, 1898; past president of the Malden Medical Society; for many years medical inspector in Malden schools; served on the staff of Malden Hospital; trustee of the Malden Savings Bank; died Jan. 10, aged 86, of a heart attack.

McAnally, William Jefferson ⊕ High Point, N. C.; Baltimore Medical College, 1897; past president of the Guilford County Medical Society; for many years health officer of High Point and member of the school board; veteran of World War I; served as trustee of the Agricultural and Technical College of North Carolina in Greensboro; died Jan. 5, aged 80, of cerebral hemorrhage.

McClellan, Wallace Roy, Akron, Ohio; Jefferson Medical College of Philadelphia, 1912; served during World War I; examining physician at Goodyear Tire & Rubber Company for many years; died in the Goodyear Hospital Jan. 15, aged 66, of rupture of aortic aneurysm.

McNamara, William E. ⊕ Lansing, Mich.; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1902; served as a member of the state council of health; fellow of the American College of Surgeons; past president of the Ingham County Medical Society; a founder of the People's State Bank; on the staff of the Edward W. Sparrow Hospital and St. Lawrence Hospital, where he died Jan. 19, aged 76.

Manning, Gustave Randolph ⊕ New York City; Albert-Ludwigs-Universität Medizinische Fakultät, Freiburg, Germany, 1899; professor of gastroenterology at New York Polyclinic Medical School and Hospital; member of the National Gastroenterological Association, of which he was secretary; president of the Society for the Advancement of Gastro-Enterology when it was formed in 1933; served during World War I; on the staff of the Polyclinic Hospital, where he died Dec. 1, aged 79, of arteriosclerosis.

Marchildon, John Woods ⊕ McClure, Ill.; Rush Medical College, Chicago, 1903; an Associate Fellow of the American Medical Association; member of the California Medical Association and the American Urological Association; formerly on the faculty of St. Louis University School of Medicine; died in Cape Girardeau, Mo., Jan. 29, aged 77.

Mayginnes, Patrick Henry ⊕ Tulsa, Okla.; St. Louis College of Physicians and Surgeons, 1897; an Associate Fellow of the American Medical Association; died Jan. 19, aged 82.

Miller, Arthur Weston ⊕ Riverside, Calif.; University of Nebraska College of Medicine, Omaha, 1927; fellow of the American College of Surgeons; past president of the Riverside County Medical Society; served during World War II; on the staff of the Riverside Community Hospital, where he died Dec. 28, aged 50, of coronary sclerosis.

Moore, Samuel Barlow ⊕ New York City; New York Homeopathic Medical College and Hospital, New York, 1901; affiliated with Metropolitan Hospital and Flower and Fifth Avenue Hospitals, where he was an alumnus trustee; formerly on the faculty of his alma mater; died Jan. 17, aged 74, of coronary disease.

Mueller, Emil F., Dyersville, Iowa; Northwestern University Medical School, Chicago, 1904; died in Sacred Heart Sanitarium in Milwaukee Jan. 22, aged 73, of coronary thrombosis.

Nibbelink, Benjamin ⊕ Kalamazoo, Mich.; Detroit College of Medicine, 1912; died in the Bronson Hospital Jan. 10, aged 66, of myocardial infarction.

Nussbaum, Samuel Solis, Kingston, N. Y.; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1927; medical director of the Kingston public school system; affiliated with Kingston Hospital and the Benedictine Hospital; died Nov. 15, aged 52, of coronary thrombosis.

Oliver, Augustus Albert, Paris, Tenn.; University of Nashville Medical Department, 1910; for many years county health officer; died in St. Thomas Hospital in Nashville Jan. 11, aged 69, of heart disease and a kidney infection.

Oliver, Robert Byron, Idabel, Okla.; University of Arkansas School of Medicine, Little Rock, 1906; served during World War I; died Jan. 9, aged 81.

Pallister, Stanley Wilson, Bearsville, N. Y.; New York Homeopathic Medical College and Hospital, New York, 1898; fellow of the American College of Surgeons; for many years on the staff of the Carson C. Peck Memorial Hospital in Brooklyn; died in Woodstock Jan. 6, aged 77, of acute dilatation of the heart.

Pierson, Percy Raymond ⊕ New Albany, Ind.; University of Louisville (Ky.) Medical Department, 1912; served overseas during World War I; on the staff of St. Edward Hospital; died in St. Joseph Infirmary, Louisville, Ky., Dec. 29, aged 65, of carcinoma of the lung.

Piper, Charles Leslie ✠ Vernal, Utah; University of Oklahoma School of Medicine, Oklahoma City, 1915; secretary of the Uintah Basin Medical Society; served during World War I; affiliated with Uintah County Hospital; died Dec. 21, aged 64, of coronary occlusion.

Proctor, Carter Atwater ✠ Sullivan, Mo.; Washington University School of Medicine, St. Louis, 1912; member of the American Academy of General Practice; died Dec. 29, aged 65, of coronary disease.

Richards, Thomas ✠ Glassport, Pa.; Jefferson Medical College of Philadelphia, 1926; president of the board of health; director of the Bank of Glassport; served on the staff of the McKeesport (Pa.) Hospital; died Dec. 30, aged 53, of coronary occlusion.

Robinson, Joseph Arnold ✠ New Bethlehem, Pa.; University of Pittsburgh School of Medicine, 1913; died in Western Pennsylvania Hospital, Pittsburgh, Nov. 26, aged 61, of heart failure.

Rodman, Nathaniel Fulford ✠ Norfolk, Va.; Jefferson Medical College of Philadelphia, 1914; member of the Southeastern Surgical Congress and the American Academy of General Practice; past president of the Norfolk County Medical Society; served during World War I; affiliated with Norfolk General, De Paul, and Leigh Memorial hospitals; died Jan. 14, aged 63, of multiple cerebral thrombosis and hypertension.

Rogers, Earl Bertram, El Paso, Texas; Northwestern University Medical School, Chicago, 1902; past president of the El Paso County Medical Society; affiliated with Hôtel Dieu, Sisters' Hospital; died in Southwestern General Hospital Dec. 31, aged 79, of carcinoma of the bladder.

Schilling, John Gaillard ✠ Houston, Texas; University of Texas School of Medicine, Galveston, 1909; fellow of the American College of Surgeons; on the consulting staff, San Jacinto Memorial Hospital in Baytown; on the surgical staff, Methodist Hospital; on the gynecologic staff, Memorial Hospital; on the visiting staff, St. Joseph's Infirmary; died Jan. 4, aged 68.

Scouller, John Samuel ✠ Philadelphia; Jefferson Medical College of Philadelphia, 1919; on the staff of the Northeastern Hospital; died Jan. 5, aged 60, of a heart attack.

Sinkler, Francis Wharton ✠ Bryn Mawr, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1900; member of the American Neurological Association; past president of the Aid Association of the Philadelphia County Medical Society; formerly on the faculty of the Graduate School of Medicine, University of Pennsylvania, Philadelphia; served on the staff of the Episcopal Hospital in Philadelphia; died in Bryn Mawr Hospital Jan. 2, aged 76, after a fracture of his hip.

Sinkler, Wharton Jr., ✠ Philadelphia; University of Pennsylvania School of Medicine, Philadelphia, 1940; on the board and formerly chief resident physician at the Hospital of the Protestant Episcopal Church; served as resident at the Hospital of the Woman's Medical College of Pennsylvania; served during World War II; died Dec. 25, aged 39.

Sorensen, Jens, Detroit; Københavns Universitet Laegevidenskabelige Fakultet, Denmark, 1888; died Jan. 18, aged 86, of coronary occlusion.

Southwick, George Edwin, Chatham, Ill.; Miami Medical College, Cincinnati, 1892; died in the Memorial Hospital, Springfield, Jan. 19, aged 85.

Stewart, J. Sewell ✠ Pittsburgh; Hahnemann Medical College and Hospital of Philadelphia, 1919; served on the staff of Shadyside Hospital, where he died Jan. 19, aged 75, of a central nervous system lesion, type undetermined.

Taphorn, D. Henry ✠ Effingham, Ill.; Washington University School of Medicine, St. Louis, 1898; an Associate Fellow of the American Medical Association; for many years county coroner; served on the Effingham city council as commissioner of health and safety from 1923 to 1931; railroad surgeon for the Illinois Central and Pennsylvania railroads; died in St. Anthony's Hospital Jan. 14, aged 82, of myocarditis and intestinal obstruction.

Tomkins, Harry S., Los Angeles; Chicago College of Medicine and Surgery, 1917; died in Duarte Dec. 24, aged 67, of carcinoma of the esophagus.

Urevitz, Abraham ✠ New York City; University College of Medicine, Richmond, 1910; member of the Medical Society of New Jersey; served during World War I; affiliated with the Hospital for Joint Diseases; died in Union City, N. J., Jan. 13, aged 67, of coronary thrombosis.

Verta, John Camillo ✠ Schenectady, N. Y.; Regia Università di Napoli Facoltà di Medicina e Chirurgia, Italy, 1893; on the courtesy staff of the Ellis Hospital, where he died Oct. 2, aged 87, of pernicious anemia.

Waters, La Verne Francis ✠ Medina, N. Y.; University of Buffalo School of Medicine, 1908; served as county coroner and as health officer; on the staff of the Medina Memorial Hospital; died in the Buffalo General Hospital Jan. 13, aged 69, of embolism after an operation for hernia.

Weiss, Solomon A. ✠ Chicago; Chicago College of Medicine and Surgery, 1914; on the staff of the Belmont Hospital; died in St. Anne's Hospital Jan. 21, aged 63, of melanosarcoma.

Worden, Rollin Duane, Ravenna, Ohio; University of Vermont College of Medicine, Burlington, 1915; served during World War I; formerly health commissioner of Portage County; during World War II served as a member of the selective service board and later as its medical examiner; died in the Lakeside Hospital in Cleveland Dec. 10, aged 64.

Yavorsky, George William ✠ Belle Plaine, Iowa; State University of Iowa College of Medicine, Iowa City, 1905; affiliated with Mercy and St. Luke's hospitals in Cedar Rapids; surgeon for the Chicago and Northwestern Railway; died in Cedar Rapids Jan. 7, aged 70, after pneumonectomy for bronchogenic carcinoma of the left lung.

Young, Roland Caldwell ✠ Toledo, Ohio; Rush Medical College, Chicago, 1921; president of the board of trustees of the Toledo Y. M. C. A.; affiliated with Flower Hospital, where he was past chief of staff, and a trustee, and Maumee Valley Hospital, where he was past chief of staff, Toledo, Mercy, and Riverside hospitals; consultant at Toledo State Hospital; died Jan. 7, aged 57, of coronary thrombosis.

DIED WHILE IN MILITARY SERVICE

Shorr, Harold Michael ✠ New York City; New York University College of Medicine, 1941; served as assistant professor of clinical medicine at his alma mater; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; affiliated with Bellevue and University hospitals; major, Medical Corps, U. S. Army; died in Camp Rucker, Ala., Dec. 16, aged 38.

Marvin, Theodore Richard ✠ Sarasota, Fla.; born in Jefferson, Ohio, Nov. 21, 1916; Ohio State University College of Medicine, Columbus, 1943; interned at the Naval and Southern Baptist hospitals in New Orleans; member of the Medical Association of the State of Alabama; initially appointed an ensign in the U. S. Naval Reserve in 1942; resigned this commission in May 1943 to participate in the Navy's V-12 medical training program; appointed a lieutenant (j. g.) in the Naval Medical Corps; became a lieutenant (j. g.) in 1944 and was promoted to lieutenant in the reserve in 1946; during World War II served in the Pacific in the USS LST-711, at Fleet Hospital no. 111 and Naval Base Hospital no. 18 and with Escort Division 7; released to inactive duty in June, 1946; became associated with the staff of the Perry County Hospital in Marion, Ala.; recalled to active duty in January, 1953, and ordered to the Naval School of Aviation Medicine, Pensacola, Fla.; designated a naval flight surgeon on completion of this course in June, 1953; ordered to the Marine Corps Air Station, Cherry Point, N. C.; last duty assignment was the Marine Air Group 11; died Dec. 30, aged 37, of injuries received in an automobile accident near Atsugi, Japan.

FOREIGN LETTERS

AUSTRALIA

Australia's National Health Service.—Until the establishment of the Commonwealth of Australia in 1901, all legislative powers resided with the six states (Queensland, New South Wales, Western Australia, Victoria, Tasmania, and South Australia), but, on federation, defense, mail and telegraphic service, and customs and excise rights were surrendered to the Commonwealth. The only health powers, however, that the states surrendered were those relating to quarantine. It was not until 1947 that the Commonwealth government obtained the power to make laws with respect to the provision of maternity allowances; widows' pensions; child endowment; unemployment; pharmaceutical, sickness, and hospital benefits; medical and dental services (but not so as to authorize any form of civil conscription); benefits to students; and family allowances. These powers were sought because three years previously the Labor government then in power had passed a Pharmaceutical Benefits Act designed to supply free to patients compounded and uncompounded drugs included in a government formulary, but the medical profession objected to the restrictions imposed by that formulary and other unacceptable provisions of the scheme. When the legislation was challenged by the Medical Society of Victoria, the High Court of Australia (corresponding to the United States Supreme Court) declared the legislation to be outside the powers of the Commonwealth parliament. In 1948, under the increased powers obtained by referendum, the Labor government passed similar legislation, but again the medical profession refused to participate. Subsequently, when the government attempted, under amended legislation, to use coercive powers, the High Court was again approached and it held that such coercion entailed a measure of civil conscription and the enactment was, therefore, invalid. There the matter remained until 1950.

Although the government in 1948 passed a National Health Service Act, under which it would have been possible to establish a complete national medical service free to the patient and paid for out of social service taxation, no action was taken under the legislation. In 1949 the Labor government was defeated and succeeded by a coalition government of Liberal (conservative) and Country (farm) party members led by the present prime minister. Sir Earle Page, a physician, was appointed Minister for Health, and soon after he announced that the government would encourage voluntary health insurance, using its funds to amplify the coverage against sickness provided by approved insurance organizations. He has received the cooperation of the medical and pharmaceutical professions in developing his plans. The following steps have been taken:

1. By arrangement with the state governments each school child under 12 years of age receives free one-third of a pint of milk per day, and in remote areas where fresh milk is not available powdered milk is supplied.

2. In July, 1950, a scheme of tuberculosis allowances was brought into operation and sufferers from the disease, while undergoing treatment, are paid cash allowances. The Commonwealth government also contributes to the cost of mass x-ray surveys conducted by state health departments and to the capital costs of construction of sanatoriums.

3. In September, 1950, a substantial list of lifesaving and disease-preventing drugs was made available free to all citizens. They are supplied to patients by approved druggists on the prescription of a physician, and the government pays the druggist. Included in the list are the sulfonamides, insulin, penicillin, streptomycin, chloramphenicol, chlortetracycline, vaccines, serums, and many other drugs of proved value, and additions to the list are made from time to time on the recommendation of a pharmaceutical benefits advisory committee, on which the medical and pharmaceutical professions are represented.

4. In February, 1951, a free medical service for aged, invalid, and widowed pensioners, and their dependents, including about 600,000 persons, was introduced. Under this service beneficiaries receive free of cost from participating physicians, any physician being eligible to participate, those services customarily rendered by a general practitioner in his office or in the patient's home. In addition to the drugs available free to all citizens, they may receive, on prescription and without cost, any drug in the British Pharmacopoeia, either alone or in combination. There is no contract between the government and the physician, but by arrangement with the Federal Council of the British Medical Association in Australia, those rendering service charge concessional rates (9 shillings a consultation and 11 shillings a domiciliary visit or about 60% of private charges). Payment is made to the doctor by the health department on production of vouchers signed by the patient, who certifies that he has received service and requests that payment be made to the doctor by the government on his behalf.

5. In July, 1953, a medical benefits scheme was started, under which the amounts payable in return for premiums by organizations offering insurance against the costs of medical care are supplemented by subsidy from government funds paid through those organizations. Commercial insurers are excluded from the scheme, and only mutual nonprofit organizations are approved, one of the conditions of approval being that the insurance organization must pay to its contributors an amount not less than the government subsidy as set out in a table of subsidies, but in no case shall the total benefit exceed 90% of the physician's account. In that way a personal obligation to meet part of the cost of medical care is imposed on the contributor, thus safeguarding available funds against abuse. As an additional safeguard payment is made by a benefit organization only on production of a physician's receipt, except in cases of hardship, when benefits may be paid before the patient has paid his physician. The amount of the subsidy varies in relation to the nature of the medical service, but for any particular service it remains the same whatever actual fee the physician charges, whether the service is rendered by a general practitioner or a specialist. The table of subsidies lists nearly 1,000 items covering the whole range of medical practice, including consultations, operations, obstetrics, radiological service, and pathological study.

For about 10 years benefits have been paid by the Commonwealth at the rate of 8 shillings a day to all persons occupying hospital beds, but voluntary insurance against hospital costs is now being supported by the payment of an additional 4 shillings a day to members of hospital benefit organizations, usually, in fact, the same organizations that offer coverage against the costs of medical care.

Because the present government did not have a senate majority until May, 1953, it hesitated to introduce new legislation that a hostile upper house would reject. Consequently the foregoing plans were developed by the promulgation of regulations under existing legislation—the Pharmaceutical Benefits Act and the National Health Service Act of 1948—but in December, 1953, the National Health Act was passed. That measure has established a firm legal basis for the services now in existence.

It will be seen that, unlike Great Britain on the one hand with a nationalized medical service and the United States on the other hand with its predilection for personal responsibility, Australia has adopted a middle course, a system of self-help aided by the resources of government. The last six months have shown that the public reaction is favorable to the proposals, as during that time there has been a vast increase in membership of insurance organizations, and already at least half of the Australian population is covered by voluntary insurance against some part of the costs of illness.

The items in these letters are contributed by regular correspondents in the various foreign countries.

ECUADOR

Hemodynamic Changes in Anemia.—Drs. Lanari, A. García, and C. Nuñez of Guayaquil reported on the hemodynamic changes that occurred in a group of men with tropical anemia of various degrees of intensity, but without cardiac, pulmonary, or infectious diseases. The quantity of hemoglobin in the blood of all patients varied between 1.7 and 8 gm. per 100 cc. The authors reported the following observations: 1. The blood volume of these patients was diminished in proportion to the quantity of hemoglobin. This diminution was of moderate intensity when the hemoglobin level was more than 6 gm. per 100 cc. It was marked when the quantity of hemoglobin was less than this. The lower the quantity of hemoglobin, the greater the diminution in blood volume. When the hemoglobin fell to 1.7 gm. per 100 cc., the blood volume diminished to about 25% of normal. 2. The amount of oxygen in the arterial and venous blood in these patients was below normal. When the hemoglobin was 8 gm. or more per 100 cc., the saturation of arterial blood was 100%. When the hemoglobin was less than 8 gm. per 100 cc., the blood was not saturated with oxygen. The degree of saturation decreased as the hemoglobin diminished down to 6 gm. per 100 cc. At this figure, saturation was about 80%. Further diminution of hemoglobin did not decrease the degree of saturation. 3. The so-called "cardiac index" increased in proportion to the diminution of hemoglobin. Thus, an index of 5.5 when the hemoglobin was 8 gm. per 100 cc. increased up to 9.8 when the hemoglobin fell to 1.7 gm. per 100 cc. 4. The velocity of circulation increased with the diminution of hemoglobin. The normal velocity was doubled when the hemoglobin dropped to 1.7 gm. per 100 cc. 5. The pulmonary ventilation in these patients was increased but this may normally show variations due to the emotional condition of the patients during the examinations. Pulmonary hyperventilation was marked when these patients were subjected to physical exercises. This hyperventilation was greater in patients with hemoglobin levels less than normal, and in such patients it lasted longer after physical exercises. 6. The pulse and respiratory rate were increased when the hemoglobin fell below 8 gm. per 100 cc. Thus, a pulse rate of 80 when the hemoglobin was 8 gm. per 100 cc. may increase to 120 when the hemoglobin falls to 1.7 gm. per 100 cc. 7. The minute volume of circulation increased as the hemoglobin decreased. When the hemoglobin fell to 1.7 gm. per 100 cc., the minute volume was triple the normal figure and it increased to even six times normal after physical exercise. 8. The volume of blood expelled from the heart with each systole was also increased and increased still more after physical exercise in these patients. (*Gac. méd. Guayaquil* **8**:397, 1953).

Blood Groups in Indians of Ecuador.—Before the Spanish Conquest, the Indian population of the coastal, Andean, and Amazonian regions of Ecuador consisted of several races that have now almost disappeared. Only a few groups of descendants of two of the coastal and three of the Amazonian races are still found, whereas the Indian population from ancient Andean races is very large, especially in the high plateaus of the Andes. The Indian population comprises 16% of the total population of the country. In a racial sense, the Indians are isolated from the rest of the population. They live in zones 10,000 ft. or more above sea level and do not mix with the white population. The contrary is observed in cities of the lower zones, where mestizos predominate as a result of several generations of marriages between the Spaniards and Indians. Dr. A. Santiana, professor of anthropology of the Central University of Ecuador, carried on extensive researches on the blood groups in Indians. Of 8,112 Andean Indians, 7,707 (95.01%) had blood that corresponded to group O; 290 (3.57%), to group A; 94 (1.16%), to group B; and 21 (0.26%), to group AB. Of 1,055 Amazonian Indians, 1,036 (98.20%) had blood that corresponded to group O; 17 (1.61%), to group A; 2 (0.19%), to group B; and one, to group AB. Of 150 coastal Indians, who are probably of the same somatic Andean type, 100% corresponded to group O. The results were probably due to the small number of Indians in this group. Probably Indians of groups A and B could have been found in a study of larger groups of coastal Indians.

Antihistaminic Agents for Burns.—The antihistaminic agents have local anesthetic properties. Some antihistaminics are more effective than procaine. These substances, used in the prevention and in treatment of solar erythema and of erythema due to ultraviolet radiations, give good results. One of the antihistaminic agents Dr. P. Naranjo used in treating burns due to heat and chemical substances was pyrilamine (Neo-Antergan). He applied a solution of 0.1 to 0.5% of the antihistaminic agent over the burn every 15 or 30 minutes until the pain was relieved. Then he applied an antihistaminic salve daily for one to three days. The pain was usually controlled within 30 minutes after the first application of the solution. Thereafter only a slight pain was reported by the patients. When the antihistaminic agents are applied in the first minutes after the burn, blisters and edema of the skin are prevented. If blisters are already formed, they reabsorb within 24 hours. If secondary infection occurs, the well-established treatments for infection should be used (*Gac. méd. Guayaquil* **8**:414, 1953).

FINLAND

Campaign Against Tuberculosis.—With a population of 4 million, Finland has only 13.5 inhabitants per square kilometer. This thin distribution of the population partly explains some of the features peculiar to the behavior of tuberculosis in the country, but on the whole the campaign against this disease has followed a pattern closely resembling that of other European countries, especially Denmark, Norway, and Sweden. As in these countries, voluntary activities have gradually been taken over by the state and local authorities in an attempt to secure more effective centralization. In 1944 a state tuberculosis committee was appointed to draft new legislation for the control of this disease. The recommendations of this committee led to a new law, which became operative in 1949 and provided for the partition of the country in 19 tuberculosis districts under the administration of the Ministry of Health. Each such district is required to maintain a tuberculosis bureau and a sanatorium. Special institutions must be provided for occupational therapy, for children threatened by tuberculosis in their homes, and for refractory patients. BCG vaccination is not compulsory, but everyone who has reached the age of 15 must submit to a medical examination at his local bureau unless he can present a medical certificate, not more than three months old, by a lung specialist exempting him from examination. BCG vaccination was started in 1941, but during the next few years little use was made of it, and only about 25,000 persons had been thus vaccinated up to 1944. In 1951 a total of 123,063 persons were vaccinated with BCG, and the total number of such vaccinations has risen to about one million. In spite of this vaccination being voluntary, nearly every newborn infant is now vaccinated with BCG, the reaction being subsequently tested with tuberculin at a tuberculosis bureau. Tuberculin testing is repeated at the time of entering school, and BCG vaccination is repeated when necessary. Since 1943 almost every tuberculin-negative man called up for military service has been vaccinated with BCG provided by a bacteriological laboratory in Gothenburg. The Tuberculosis Association, founded in 1941 by the patients themselves, has grown into a powerful organization with about 30,000 members and both the will and the means to give the victims of tuberculosis substantial help. In 1953, this association opened a high school with accommodation for 52 pupils. A center for the reeducation of tuberculous war invalids has also been provided by this association.

Crude Liver Extracts for Cirrhosis of the Liver.—The work of Hoagland, Ralli, and others in the United States on cirrhosis of the liver treated by intravenous injections of crude liver extracts has been closely followed in Finland where two studies of this problem have been undertaken by Dr. Gustaf Østling and Dr. Erik Adlercreutz respectively. The first tests were not as encouraging as the more recent ones. A crude liver extract called neoheptomin did not at first contain much vitamin B_{12} but the later batches contain a large amount of this vitamin. Both these studies are published in *Nordisk medicin* for Nov. 26, 1953, and although Østling's is chiefly concerned with general principles and a review of the literature, the study by Adlercreutz deals

with concrete experiences in Helsingfors, where about 30 patients with hepatic disease have been given this treatment. He is chiefly concerned with 10 of these patients in whom the response to intravenous injections of neoheptomin was most instructive. Eight of these patients had Laennec's cirrhosis. Adlercreutz notes that admission to a hospital, with its attendant rest and dietetic treatment, often by itself effects great improvement and so obscures the issue. In 8 of the 10 patients, however, the marked diuresis that occurred soon after the intravenous injections were started pointed to their being effective in themselves. In one patient ascitic fluid disappeared completely, but the most striking effect was the improvement in the patient's general condition. In two patients the injections had to be discontinued for a week or more because of fever, but after this interval the treatment was resumed without any further complications. To avoid a sense of heat in the face during the injections it was found desirable to give them very slowly. Adlercreutz was puzzled over the character of the active principle in crude liver extracts, and he has found no great difference of potency in the original neoheptomin, which was poor in vitamin B_{12}, and that of the later batches, which contained more of this vitamin. He also could find no marked difference of effect resulting from varying the size of the doses. This treatment may prove to be a valuable auxiliary to preoperative and postoperative treatment. In his more recent cases Adlercreutz has found it desirable to reduce the chances of a violent reaction by giving neoheptomin by drop infusion with 100 to 200 ml. of a glucose solution.

ISRAEL

Side-Effects of Penicillin.—In the fall of 1953 in a four week period 13 cases of cutaneous reaction due to penicillin were observed by I. Katzenellenbogen of the Central Department for Skin Diseases of the Workers' Sick Fund in Jerusalem, as reported in the *Harefuah*, Dec. 15, 1953. Seven men and six women aged 17 to 47 were treated. All but two previously had received penicillin; four by injection and seven by ointments and lozenges. In three, papulovesicular eruptions developed on the palms, soles, and genitocrural regions. The eruption appeared 24 to 36 hours after the first injection. Three patients presented a generalized urticaria, appearing between 7 and 10 days after penicillin treatment and lasting from 7 to 14 days. In seven patients a combination of an immediate and delayed reaction occurred, beginning a day or two after the injection with a delayed serum sickness developing 10 to 17 days later. In one patient, with no previous history of fungous disease, a severe papulovesicular rash appeared on the palms, soles, and axillary and genitocrural regions and was preceded by perlèche and an erythematous rash on the chin. No fungus was found either microscopically or by culture. Intradermal tests of sensitivity to penicillin and trichophytin in this case were positive. In two patients the eruption occurred after a single injection, the local reaction appearing after 24 hours and generalized urticaria with angioneurotic edema 17 days after the injection. In four patients otolithic vertigo was observed with alteration of the caloric reaction. In one patient with a severe delayed serum sickness cortisone relieved the polyarthritis, the high fever, and the urticarial rash, without influencing the vertigo. In the same patient an eruption developed on previously tanned parts of the body.

Seven of 11 intradermal tests with 2,000 units of crystalline penicillin were positive, but patch tests with procaine penicillin and crystalline penicillin were negative in all patients. Only 2 of 11 tests with trichophytin were positive. The Prausnitz-Kustner reaction was positive in one of three patients tested. Dermographism was observed in all urticarial cases; hyperhidrosis, only in two.

Possible causes of so many penicillin reactions in a short period include (1) previous or currently existing mycosis, (2) indiscriminate use of penicillin ointment, lozenges, etc., (3) the allergic condition of the patient, and (4) the brand of the drug. The author recommends avoiding the local use of penicillin or its injection without proper indication. It is suggested that in suspected cases antimycotic and antihistaminic treatment be given at the same time as the penicillin injections. Cortisone was helpful in severe cases of delayed serum sickness.

Scholarships for Medical Students.—The central committee of the Medical Association proposes to maintain 10 scholarships in the amount of 300 Israeli pounds per annum each, out of contributions from physicians all over the country. This undertaking was prompted by both the difficult economic position of the students already studying in institutions of higher learning and the fact that the number of young physicians is extremely small compared with that of older ones. In several branches of medicine, there is a danger that in 5 or 10 years a patient may have to wait several months for a tonsillectomy or an orthopedic operation. Members of the Workers' Sick Fund have already been queing up six months or longer for such operations. At the end of 1952 about 48% of all physicians in the country were over 52 years old, while the number of physicians under 36 years of age did not exceed 12%. The position is particularly acute in specialized branches; about 65% of the dermatologists, 60% of the radiologists, 62% of the ophthalmologists, 52% of the psychiatrists and neurologists, 50% of the otolaryngologists, 54% of the gynecologists, 52% of the pediatricians, and 51% of the internists are 50 years old or over. Only 8% of the otolaryngologists, 10% of the dermatologists, 11.5% of the radiologists, 11% of the ophthalmologists, 19% of the psychiatrists and neurologists, 22% of the pediatricians (mostly women), 16% of the gynecologists, 17% of the internists, 23% of the general practitioners, and 25% of the surgeons are under 40 years of age.

Since the establishment of the state of Israel in 1948, the population of the country has more than doubled, but owing to the composition of the mass immigration, which has derived chiefly from depressed countries, only 1,008 physicians have entered the country during this period. These now constitute 29.4% of the total number of physicians in Israel. Only 106 physicians have come from Egypt, Tunis, and other parts of Africa. It is encouraging, however, that most of the immigrating physicians have been young. Of the 684 physicians in the country who are not more than 41 years of age, 587 immigrated to Israel after the establishment of the state.

TURKEY

Idiopathic Anemia.—In *Dirim*, vol. 28, no. 3, Prof. Zeki Hakki Pamir and Dr. Cavit Sökmen described a case of idiopathic aplastic anemia. Of 4,826 patients admitted to Professor Pamir's clinic from 1946 to 1952, 228 had blood diseases. This first patient with idiopathic anemia seen in the clinic was a 17-year-old girl who, on admission, was very pale and complained of weakness, headache, palpitation, and bleeding of the gums. Following injury, external hemorrhage was arrested with difficulty. The patient weighed 56 kg., had a regular pulse rate of 92, and a blood pressure of 105/75 mm. Hg. Her liver and spleen were soft and of normal size. She had a cardiac souffle, bleeding of the gums, and ecchymotic spots on the skin. Her gastric secretion was normal. Her parents and three siblings were in good health. The coagulation time was 8 minutes, the bleeding time 30 minutes, the erythrocyte count 1,540,000, and the leukocyte count 3,000 per cubic millimeter, with 71% lymphocytes, 25% immature forms, 2% eosinophils, and 2% monocytes. The hemoglobin level was 5.5 gm. per 100 ml.; the thrombocyte count was 11,620; and there were 1.16% reticulocytes. The myelogram showed 45% lymphocytes, 18% normoblasts, 9% myeloblasts, 9% transitional cells, 8% monocytes, 5% macroblasts, 4% immature forms, and 2% reticulocytes. Results of the epinephrine test for hypersplenism and the blood plasmodium, Wassermann, and urobilinogen tests were normal. Results of the bilirubin test of the blood by the direct method were normal and by the indirect method revealed 0.04 mg. per 100 cc. Iron and liver extracts, vitamin B_{12}, folic acid, and cortisone did not benefit the patient who during her 110 days in the hospital was given seven blood transfusions. Following a transfusion the erythrocyte count increased to about 2 million cells per cubic millimeter, but decreased again after one week. The patient refused splenectomy and left the hospital.

CORRESPONDENCE

DECELERATION AND THE MOTOR CAR

To the Editor:—In this age of increasingly rapid transportation, the one hope of survival is the control of deceleration. In a paper appearing in 1946 Hugh DeHaven stated, "Up to the present, aircraft have been built solely to fly, not to crash. This single approach and design has been costly in the past and is no longer acceptable in military aircraft where a few pounds of extra weight to give crash protection has been an important concession." The modern military aircraft is built around a safety island. The wings, tail, and motor may all separate but this island remains intact and will resist impacts beyond 40 G. In this island the personnel are fastened with harness and belts designed to withstand 40 G. or more. Personnel survival in airplanes so constructed is many times greater than in the type of planes flown in World War II.

On our highways, too, we must learn to control rapid deceleration. As the speeds of motor cars have increased we have progressed through brakes on the rear wheels, four wheel brakes, and now we have reached the point of diminishing returns as far as deceleration is concerned. I have heard two motorists state, "You sure have to be careful with these power brakes, I threw my wife into the windshield twice until I learned to use them." In other words, the potential stopping power of power brakes cannot be utilized because the motor car manufacturers have not installed devices to keep the passengers in place when modern brakes are used. Where the aviation industry has met the problem with boldness and originality, the motor car manufacturers have done almost nothing to protect the persons riding in motor cars when they must decelerate rapidly. We have lost between 30,000 and 40,000 lives on our highways each year since 1915, and the number of injuries, ranging from minor to the most disabling, number over one and one-half million each year. Is this inevitable? Are we who made the machine necessarily a victim? Let us consider this problem a moment. When a motor car hits a pedestrian, that victim is hurt, his injury is immediate. The traffic engineers must increasingly separate the streams of motor traffic from pedestrian traffic just as we have at long last separated motor traffic from the railroad train. On the other hand, when two motor cars collide or when a motor car meets a fixed obstruction, no one is hurt. The bridge abutment is dented and the front of the motor car is bashed in, but the safety island in the center of the car is intact, no one is hurt. The forces have all been applied to inanimate objects. Not until the occupants began slamming around inside the car or hurling themselves against objects outside the car do they receive injury. Can these injuries be prevented? In accidents of comparable degree affecting modern military aircraft, the personnel walk away with hardly a scratch. In stock car races the drivers put their cars into worse spills than often occur on the highway, and regularly they step out of the car, wave to the crowd, and walk away. Mr. William W. Harper describes it in phrases of the utmost clarity: "Let us assume that a vehicle collides with a solid fixed object at a speed of 30 miles per hour. Let us assume further that the car is crushed in a distance of two feet. This means that the velocity of the vehicle has decelerated from 30 miles per hour down to zero in a distance of two feet. This represents a deceleration rate of 483 feet per second per second. Such a deceleration is 15 times the acceleration of gravity, which is 32.2 feet per second per second. This unit of gravity is called the G. For convenience, we say that the vehicle suffered a 15 G. crash. But how does the vehicle occupant behave in such a crash? At the moment of impact he has the same velocity as the vehicle. As the vehicle crashes to a full stop he continues forward at almost the same speed of 30 miles per hour and collides with the dash and windshield. By the time his body reaches those objects they are at rest, or nearly so. Assuming that the combined crushing of his body and vehicle interior will reduce his velocity to zero in a distance of two inches, he will have suffered a deceleration of almost 5,700 feet per second per second, or 176 G. Although the occupant might have survived without injury the 15 G. crash of the vehicle, he cannot escape injury or death from the 176 G. crash of his body against the interior of the vehicle. The physics of injury tells us something which seems paradoxical: If the occupant wore the car, as he would a suit of armor, the crushing of the car exterior in a collision would absorb tremendous amounts of impact energy and protect him from bodily injury. The occupant would be spared injury unless his passenger space became extensively crushed. But for some unexplained reason the teachings of physics have never been understood or accepted by the motorist—so, rather than 'strap on' the vehicle and take advantage of it's protective armor in a crash, the motorist watches the vehicle crash relatively slowly to a stop and then dashes himself violently to pieces against its interior! This makes no sense at all, but it is still standard practice after 50 years of automotive accident history."

The Colorado State Medical Society adopted the following resolution and sent it to all the motor car manufacturers in the United States and to all the other state medical societies urging them to make similiar resolutions: "Whereas motor car deaths in the United States of America number between 35,000 and 40,000 annually and motor car injuries number about one and a half million annually; and whereas there seems little likelihood of any great reduction of motor accidents in the near future; and whereas studies by physicians and physicists have clearly shown that motor injuries and motor deaths can be strikingly reduced by the use of safety belts and safety shoulder straps, therefore be it resolved: That the Colorado State Medical Society will give all possible aid to those measures which will reduce the frightful mortality and injury rate resulting from the use of motor cars, and be it further resolved: That the Society hereby recommends to the motor car manufacturers of America that they equip all automobiles with safety belts to meet the specifications of the C. A. A. Technical Standard Order, T. S. O.-C22 A, November 15, 1950, and further recommends that these manufacturers provide seats, cushions, and doors which will withstand impacts of 10 to 15 G. without injuries."

Poliomyelitis took 3,300 lives in 1952. The motor car took 10 times as many. Poliomyelitis injured 14,000 persons in 1952. The motor car injured 100 times as many. Our motor car manufacturers must build the automobile to crash. An active program resulting in safety belts for every motor car occupant can save more lives per dollar than anything else we can do.

HORACE E. CAMPBELL, M.D.
Chairman, Automotive Safety Committee
Colorado State Medical Society
835 Republic Building
Denver.

CHLOROPHYLL

To the Editor:—I wish to reply to several points raised in this section, Oct. 24, 1953, dealing with chlorophyll. These points stemmed in part from my earlier letter on this subject in the June 27, 1953, and in part from an editorial in the Aug. 29, 1953, issues of THE JOURNAL.

I paid commercial chlorophyll derivatives the compliment of being pheophytins or pheophorbides. This placed them nearer in structure to chlorophyll than they actually are. It now develops that in reality they consist of chlorin e and rhodin g. These compounds are more broken down than pheophorbides in that one of the five rings, which constitute the normal structure of chlorophyll, has been opened. In this, by the way, Dr. Westcott was misled when he said that "The removal of magnesium from 'chlorophyllin' does not lead to pheophorbides but to chlorins"; the removal of magnesium does lead to pheophorbides and the chlorin results only from an additional breakdown reaction by alkali (Fischer and Stern, 1940). The commercial "derivatives," therefore, have had removed from them the magnesium, the phytol, and the methyl alcohol; in addition one ring has been broken and an atom

of copper has been added. It follows that the term chlorophyllin, still less chlorophyll, should no longer be applied to these materials. They should be called what they are, namely copper chlorins. It is as though one were to split the molecule of vitamin B into its two constituent parts, break off the side-chain, substitute the sulfur atom for something else, and then market the material as "vitamin B derivative." I think most nutritionists would agree that such nomenclature would be misleading and dangerous.

Dr. Langley says I ignored a paper he presented at the Federation meetings in April. My letter was written on Feb. 16. I now have certain criticisms of his paper; they are as follows. In the first place, hydrogen sulfide-air-water mixtures are notoriously unstable and are oxidized to sulfur at a moderate rate merely on standing; the reaction is probably catalyzed by heavy metal ions, so that rigid controls would needed to establish an effect due to copper chlorin. In the second place, the copper chlorin would contain enough copper to react chemically with an appreciable fraction of the hydrogen sulfide. Langley's data suggest that about 0.25 mM. of hydrogen sulfide reacted with 30 mg. of copper chlorin, the copper content of which is not stated. In the third place, no mention is made of comparative tests on other copper-containing compounds or on other catalysts; ferric chloride, for instance, is very active in this oxidation. Last, it does not seem probable that the oxidation of hydrogen sulfide has any close relationship to the deodorization of organic bases, such as skatole, that are not readily oxidized and contain no reactive sulfhydryl group.

These considerations raise the problem of what deodorization really means, in a biochemical sense. Odors that are considered disagreeable may arise from substances of extremely varied chemical constitution. Some of these can be oxidized, some can perhaps be adsorbed on active surfaces, and some can perhaps be precipitated or removed in other ways. A given substance, therefore, might well deodorize chemicals of a certain type and yet be quite unable to affect others. The idea of a general deodorant is difficult to justify since no general reaction is involved.

I must say that it is interesting that responsible workers such as Harrisson and Langley are now trying to obtain evidence that copper chlorin does actually deodorize. It would have been better to have done this first, before putting the material on the market. Usually research on the value of a therapeutic agent is done before, rather than after, its sale to the public.

KENNETH V. THIMANN, PH.D.
Professor of Biology
Harvard University
Cambridge 38, Mass.

HEMOPHILIA AND CLOTTING TIME

To the Editor:—The findings of Brinkhous and his associates (Newer Approaches to the Study of Hemophilia and Hemophiloid States, *J. A. M. A.* **154:**481 [Feb. 6] 1954) that hemophilia may range in intensity from severe to subclinical, that the clotting time may be normal in the mild type, and that the antihemophilic factor appears to remain fairly fixed in any one patient with hemophilia confirm our earlier observations, the importance of which we have repeatedly emphasized (*Pediatrics* **3:**312 [March] 1949; *Am. J. M. Sc.* **223:**401 [April] 1952). They likewise stress the difficulty of diagnosing the mild type of hemophilia. Among the tests they employ is a one stage prothrombin time of serum, a test slightly modified from the original that I developed and reported in 1947. Recently, we described a new test that we have designated as the thromboplastinogen activity time (T. A. T.). (Quick, A. J.; Stapp, W. F. and Hussey, C. V.: *J. Lab. & Clin. Med.* **39:**142 [Jan.] 1952). In a series of over 100 persons with hemophilia, we have found this test reliable, even when the disease is exceedingly mild. The procedure has the added advantage of great simplicity.

While it is the prerogative of an investigator to devise his own schemes, outlines, and classifications, one may, nevertheless, question whether a condition such as labile factor deficiency should be shifted from the category of hypoprothrombinemia and pseudohypoprothrombinemia to the hemophiloid group and be named hemophiloid state A as Brinkhous and his associates propose. In the past, it has been convenient and helpful to the clinician to divide the hemorrhagic diseases into those with a prolonged and those with a normal prothrombin time. Characteristically, hemophilia and hemophilia-like conditions have a normal, while labile factor deficiency has a prolonged prothrombin time. In any consideration of hemophilia-like states, the disease produced by a circulating anticoagulant seems to warrant not only inclusion but an important position in the classification. We have obtained fairly conclusive proof that the anticoagulant is an antithromboplastinogen and that the condition, therefore, may justifiably be designated as an antithromboplastinogenemia. The disease is important, first, because it is easily mistaken for true hemophilia and, second, because it does not respond to transfusion even of fresh blood. While the newly reported aberrant types of hemophilia are still mostly ones of academic interest, antithromboplastinogenemia presents a problem that any clinician may be confronted with unexpectedly.

ARMAND J. QUICK, M.D.
Professor of Biochemistry
Marquette University School of Medicine
Milwaukee.

SANATORIUM TREATMENT FOR TUBERCULOSIS

To the Editor:—Is sanatorium treatment for pulmonary tuberculosis necessary? The current trend in the treatment of pulmonary tuberculosis is away from the use of sanatoriums. Two hopes are responsible for this; first, that drug therapy will prove to be a specific cure for tuberculosis and, second, that bed rest is either no longer needed or can be taken as well at home. It is already quite clear that the first of these hopes is unfounded. Even the most complete program of chemotherapy plus rest, collapse, and resection has failed to control some cases. Chemotherapy plus rest alone has likewise failed in at least 10% of those persons with moderately advanced and 40% of those with far advanced disease. The question of whether chemotherapy with or without rest will prove to be curative for favorable types of tuberculosis will require several more years of observation of initially successful cases. As regards the hope that bed rest is not needed to treat pulmonary tuberculosis, a review of our knowledge of the subject is considered pertinent.

Since it was originally introduced at the turn of the century, there has been little doubt of the value of bed rest in the general management of pulmonary tuberculosis. As time passed, its practice became standard. Its use reached a peak when 24 hours a day in bed for four to eight months or more was recommended for asymptomatic, early minimal lesions. After a half century of its use, our knowledge of how bed rest works and of how much is needed in the individual case is scanty indeed. Furthermore, because of great advances in treatment, new data on unaided bed rest will not be available. From what information we have, however, we may assume that the action of bed rest is twofold—rest and the horizontal position.

Physical rest is the most obviously effective aspect of bed rest. Rest reduces movement of the diseased lung by lowering body metabolism and oxygen requirements. Rest also probably promotes nonspecific resistance against infection, thus augmenting the bacteriostatic effect of drugs. While there can be no doubt of the importance of rest, there has been disagreement over its strictness and duration. For instance, no relationship was found between duration of modified bed rest before chemotherapy and the long term behavior of minimal tuberculosis when 589 cases were analyzed retrospectively (Mitchell: *Am. Rev. Tuberc.* **67:**401, 1953). The results were superior to those observed in a similar though untreated group (Reisner and Downes: *Am. Rev. Tuberc.* **51:**393, 1945). Furthermore, in comparable groups of patients with minimal disease, De Friez and associates have found results about the same with strict as with modified bed rest. Can it be that we have prescribed excessive bed rest through lack of a more specific treatment?

Maximum rest is not always attained by spending 24 hours a day in bed. Some patients are more apt to obtain physical rest in a sitting or semireclining rather than recumbent position or by mixing periods of reclining with various mild ambulant

activities. This becomes a necessity in elderly or emphysematous patients for whom strict bed rest is frequently harmful. Rest of the mind is achieved by light reading, inconsequential conversation, pleasant contemplation, or just doing nothing. Rest of the emotions requires, in particular, the avoidance of responsibilities and personal problems. Such optimum physical, mental, and psychological rest is usually not attainable in the average home but may be found in the sheltered and simplified environment of a good sanatorium.

The second bed rest factor, the horizontal position, can, of course, be accomplished in the home as well as in the sanatorium. It has been suggested that the pulmonary circulation in the upper lungs may be virtually nil in the upright position; poor circulation adjacent to well-ventilated alveoli thus produces an alveolar environment rich in oxygen and poor in carbon dioxide, known to be favorable to the growth and multiplication of tubercle bacilli (Dock: *Am. Rev. Tuberc.* 53: 297, 1946). This concept has now been supported by Scott in animal and by Martin, Cline, and Marshall in human experimentation. The hypothesis that oxygen lack favorably influences a tuberculous lesion is supported by the observation that persistence of air-containing cavity has been associated with most failures of modern chemotherapy. Furthermore, Wright Place, and Princi have observed that there is 10 to 40% less air in the lung in the recumbent than in the erect position, making the recumbent position a minor bilateral lung collapse.

Perhaps the most vital contribution made by the good sanatorium today, and not always clearly recognized, is the education of the patient. The patient who is put to bed in a sanatorium is first given encouragement and hope; in addition, he is taught respect for a relapsing and contagious disease, a respect made necessary by the undue optimism generated, in particular, by the lay press. Day after day physician and nurse will be able to impress him, individually and in groups, with the necessity of acquiescing to his disease, at least temporarily, and, in appropriate cases, of managing his later life in accordance with his condition. Above all, the patient is impressed with the importance of taking for a long period, and without interruption, drugs that not infrequently cause unpleasant side-reactions. These reactions, whether hypersensitivity, toxicity, or intolerance, have led patients or their physicians to stop or interrupt the use of one or more drugs, thus greatly increasing the risk of loss of benefit and the emergence of drug-resistant bacilli. Unless the patient fully understands the alternative to a successful outcome of treatment, he cannot be expected always to cooperate in a prolonged, often tiresome and unpleasant routine. The time required for this education is determined more by the personality and intelligence of the patient, than by the nature and extent of the disease. As evidence of the truth of what he is taught, the patient will see around him other patients who have benefited by their training and still others who have failed to control their disease through lack of it. It is quite likely that this contact with other patients, or the knowledge gained by experience, so to speak, is the most important aspect of sanatorium treatment today. It is for these reasons that a new patient can seldom be educated as effectively in bed at home or in a physician's office, even when the physician has a thorough and up-to-date knowledge of tuberculosis.

A final but important point is segregation of an infectious case of tuberculosis, still an important public health consideration. Most new patients, when properly treated, will be noninfectious in from one to six months after starting treatment, but not all. And good cough and expectoration habits can be taught most effectively in "classes" and under continuous professional observation at the sanatorium.

There is the possibility that certain select but as yet undefined patients may not require rest because of modern chemotherapy. A 2 to 12 month period of observation may be necessary to select these favored persons. This observation may be conducted most profitably in a sanatorium. There are others whose disease is such that we know at diagnosis that chemotherapy will not do the whole job. For such patients, as well as for indigent and custodial patients, sanatorium treatment and all its adjuncts are as necessary now as ever. Whatever the prognosis seems to be, however, a period of education and orientation is important to all new patients; this is accomplished best in the sanatorium. Despite drastic improvements in treatment, the sanatorium still plays a vital part in dealing with pulmonary tuberculosis.

ROGER S. MITCHELL, M.D.
Clinical Director, Trudeau Sanatorium
Trudeau, N. Y.

INTERNATIONAL HEALTH

To the Editor:—The article "Opportunities and Responsibilities of the Physician in World Affairs" by Dr. Trimble in the Nov. 28, 1953, issue of THE JOURNAL is extremely timely and acutely points up an area of new responsibility for physicians. The promotion of health abroad has become an integral part of United States foreign policy based on the concept that poverty and mass illness are interrelated. Today the United States is spending an estimated 51 million dollars on international health programs through several agencies. Organized medicine must undertake responsibilities lest health programs be developed without benefit of the best medical knowledge.

One of the foremost deficiencies is the lack of personnel adequately trained to deal with the exotic diseases. While the underlying principles governing diagnosis, treatment, and control are similar to those of temperate zones, there are considerable differences in the relative importance of these principles. For example, most of the tropical diseases depend on arthropod vectors for transmission; nonhuman reservoirs of infection exist in animals such as bats, armadilloes, and crabs. It should be noted that a minimal knowledge of tropical medicine would raise the index of suspicion so that the physician would make early diagnosis of schistosomiasis, filariasis, kala-azar rather than allow long delays before the beginning of specific therapy and institution of preventive measures as happened in World War II. Even a few hours of instruction in our medical schools would go far in avoiding such happenings.

In addition to the assignment of physicians abroad for peacetime programs and the need for training in tropical diseases in case of national emergency, one is impressed with the increasing trade with tropical areas and the increasing amount of travel to and from these areas. These are some of the considerations that are giving a new importance to tropical diseases to physicians of the United States.

BRIG. GEN. G. R. CALLENDER
President, American Academy of Tropical Medicine
3245 Cleveland Ave., Washington, D. C.
FREDERICK J. BRADY, M.D.
President, American Society of Tropical Medicine and Hygiene
16 Woodhaven Blvd., Bethesda, Md.

TONSILS AND ADENOIDS

To the Editor:—The symposium on the tonsil and adenoid question in THE JOURNAL, Feb. 13, 1954, is a boon to those of us who cope with this problem daily and who will profit by these expert opinions. The deformity of excessively high palates with resulting nasal obstruction, although not part of this subject, is such a common basic factor in hypertrophy of the tonsils that it should be mentioned as part of the problem. Primary disease of the tonsils is not common. Tonsil enlargement is often due to neutralizing lymphatic absorption of nasal infections promoted by obstructions. In children with an allergic tendency, particularly distressing complicating symptoms are not relieved by tonsillectomy. The advantage in including nasal obstructions in this discussion is that, whether the tonsils are removed or not, when such obstruction is present early application of orthodontia procedures often lowers the nasal floor enough to separate the septum from the turbinates and give relief of symptoms.

PERCY K. TELFORD, M.D.
833 W. Mountain St.
Glendale 2, Calif.

MISCELLANY

FEDERAL COMMUNICATIONS COMMISSION RULES REGULATING THE OPERATION OF MEDICAL DIATHERMY EQUIPMENT

In a public notice of Feb. 19, 1954, the Federal Communications Commission stated that a considerable number of medical diathermy machines that do not comply with its rules are still being used by doctors, or other persons, who either are not aware that they are violating the law or do not appreciate the compelling reasons that forced the commission to adopt its present rules governing the use of medical diathermy and other types of industrial, scientific, and medical equipment.

WHY IS IT NECESSARY TO COMPLY WITH THE COMMISSION'S RULES?

Some of the harmful effects that can result from diathermy machines that radiate excessively are indicated below:

National Defense.—It has been conclusively demonstrated that both planes and missiles can be guided to their targets by following radio signals. Because of that fact, the United States has instituted a comprehensive program known as CONELRAD (CONtrol of ELectro-magnetic RADiation), which is designed to minimize the usefulness to an enemy of radio signals originating in this country. A CONELRAD program for the control of radio broadcast stations during possible air raids is already in effect. Since planes can also utilize signals emanating from excessively radiating diathermy machines, it may be necessary to put such a program into effect with respect to such equipment, particularly if equipment of this type that radiates excessively remains in use.

Interference to Safety Radio Services.—Radio is of vital importance to safety. Radio is not only used for direct communication between planes and between air and ground but as a navigational aid, particularly when visibility is limited. Radiation from diathermy equipment on frequencies outside of the assigned bands is a potential hazard to both aeronautical communication circuits and to airplane landing systems. Among other important safety services are the Police Radio Service and the Fire Radio Service. In order to avoid serious accidents and the loss of life and property, it is essential that radiation from diathermy machines be confined to assigned bands or be adequately suppressed. That means that only equipment that complies with the commission's rules should be used.

Interference to Broadcast Reception.—One common type of interference from medical diathermy equipment concerns the reception of aural and television broadcast programs. Unfortunately, many doctors, as well as other users, apparently believe that this is the only interference that their diathermy equipment is capable of causing. For the benefit of operators of medical diathermy equipment, the commission has answered the following specific questions: "Why should I worry if I have received no complaints about my equipment?" The fact that a physician or other person has not received a complaint is not a guarantee that his diathermy machine is not causing objectionable interference, perhaps many miles away. The only practical way of knowing that diathermy equipment is not causing interference is through the use of type approval or certified equipment.

"If my equipment was legal before June 30, 1953, why is it dangerous now?" Medical diathermy equipment that radiates excessively has been an interference problem for a long time. The commission's rules initially became effective June 30, 1947; however, to enable the owners of existing equipment to amortize their investments, the commission gave the owners and users of equipment manufactured and assembled before July 1, 1947, a total of six years, until June 30, 1953, within which to comply.

"What should the owners and operators of noncomplying equipment do?" The owner or operator of a medical diathermy machine that does not comply with the commission's rules should do one of the following things: (*a*) Obtain a machine that has been type-approved by the commission and that bears a type approval number. A certificate of type approval means that the commission's engineers have tested a prototype of the machine and have found that it complies with the requirements. Type approval is not an absolute guarantee that the machine will not cause interference, but it does indicate that, under normal conditions of installation and operation, the machine will not do so. (*b*) Have a competent engineer certify that the old type machine is capable of operating in accordance with the commission's rules. Such certification is accomplished in the manner specifically prescribed in the commission's rules; however, the owners of older diathermy machines are cautioned that the certification of such machines will usually require extensive modification or shielding by a skilled engineer, as well as the use of special field intensity testing equipment, and the rules also require that the certification must be renewed every three years. In many cases the certification process may prove more costly than the purchase of a new type-approved machine.

"How does the commission propose to enforce its rules?" It is hoped that when these facts are brought home to the users of illegal equipment, particularly those that relate to the dangers that may result from the use of such machines, the commission will receive a maximum degree of cooperation in the elimination of the illegal equipment; however, because of the serious potential dangers involved in the continued use of illegal equipment, the commission does not intend to rely solely on an educational campaign. It intends to use every means at its disposal to identify and eliminate the use of any equipment that does not comply with its rules.

The commission warned users of illegal equipment that the commission has available to it, and intends to follow, the following enforcement procedures wherever appropriate: (1) the issuance of cease and desist orders, enforceable in the courts, directed to the users of the illegal equipment; (2) application to the courts for injunctions against the use of illegal equipment; and (3) the institution in aggravated cases, of criminal proceedings against the operators of illegal equipment.

MILLIEQUIVALENTS

For many medical men who qualified more than a decade ago milliequivalents share some of the mystery that irrational numbers hold for the nonmathematical. But, simple units of measurement that they are, they have largely replaced such traditional though flexible units as milligrams per 100 ml. to record concentrations of ions. While readily convertible to metric units, their special features make them preferable for the estimation of the electrolytes of the plasma, which is becoming a commonplace laboratory investigation, essential for carrying out rational replacement or maintenance therapy of patients suffering from electrolyte depletion.

Milliequivalents are units of measurement no less than are the other, more familiar terms of metric weight; but they are units with this difference, that instead of being related arithmetically to other units of weight or volume within the metric system they imply a chemical relationship between the various ions under consideration. Thus, one milligram of sodium is not related to one milligram of potassium or one milligram of chloride except in so far as these amounts weigh the same, but one milliequivalent of sodium is chemically equivalent to one milliequivalent of potassium and to one milliequivalent of chloride and to one milliequivalent of bicarbonate, and in fact to one milliequivalent of any substance, although the actual weights of these amounts are not the same when expressed in milligrams. The equivalent weight of a substance is a chemical term defining that weight of the substance that will combine with or displace one gram-atom of hydrogen. A milliequivalent weight of a substance is its equivalent weight divided by 1,000, that is, the weight of the substance that will combine with or displace one milli-gram-atom of hydrogen. Adapting an old axiom, things that are equivalent to the same thing (in this case hydrogen) must be equivalent to one another, and it is this that justifies the use of the term milliequivalent in clinical chemistry today. Expressed in these terms, fluctuations in concentrations of electrolytes—not only in plasma but also in urine and gastrointestinal contents—come to have some meaning in relation to one another. Calculation of concentrations expressed in milliequivalents per liter from those in milligrams per 100 ml. is done by

Adapted from Milli-Equivalents, editorial, British Medical Journal 1: 205 (Jan. 23) 1954.

multiplying the milligrams per 100 ml. by 10 to bring the concentration to milligrams per liter, and then dividing by the equivalent weight of the ion in question. The equivalent weight of the monovalent ions sodium, potassium, and chloride is equal to the atomic weight, namely, 23, 39, and 35.5 respectively. Some laboratories report the concentration of chloride in the form of sodium chloride, in which case the concentration in milligrams per 1,000 ml. must be divided by 58.5 and not by 35.5. Special factors have been calculated for the conversion of bicarbonate from volumes per cent and protein from grams per cent to milliequivalents per liter.

BUSINESS PRACTICE

The following material is based on a public relations manual issued by the Public Relations Department of the American Medical Association.—ED.

THE PHYSICIAN AND HIS RECEPTION ROOM

Although many persons speak nostalgically of the "old family doctor"—the combination physician, father-confessor, and friend—few would entrust their care to him today, for often he had more sympathy than skill to offer. It is from fond memories of the horse-and-buggy doctor, however, that people draw their conception of the ideal physician. To the patient, a good physician is "gentle, kind, even-tempered, always available, tireless, inexhaustibly patient, everlastingly resourceful, and, up to this morning's radio broadcast, completely informed."

A good doctor-patient relationship, often termed "the most important factor in the practice of medicine," depends not only on giving people the best possible medical service but also in maintaining their confidence and friendship. Too often patients complain that doctors are cold and impersonal. The affection and friendship of the patient is won through giving him frank and friendly attention and relieving pain and easing the mind but, above all, by treating patients as persons. Patients want to feel they are important to the doctor as persons, not just as clinical cases. Says one physician, "Be glad to see patients, even if you are a little tired. Be frank; be prompt; and never give the impression that you are doing the patient a favor." Skill is no substitute for kindness. Cold, calculating science can never replace the warmth of a sympathetic personality. Healing the body is not enough. If a physician is to meet his medical responsibilities fully, he must comfort the soul, too. This is not the task of the psychiatrist alone. It is the plain duty of every practicing physician.

A patient may get his first impression of a physician as he enters the reception room. It is not necessary to hire an expensive decorator to redo the waiting room nor is it necessary to spend a great deal of money on furniture, carpeting, or drapes. The reception room should be decorated in cheerful harmonious colors, bright but not gaudy. A neat, comfortable, and attractive reception room shows patients that their doctor has their welfare at heart. In order to cut tensions produced by waiting to a minimum, the reception room should be tastefully and attractively decorated. The furniture should be comfortable and adequate for the peak number of patients to be accommodated each day. It should be arranged for reading or conversation. The room itself should be well ventilated and the temperature regulated for comfort. If possible, storage space for coats, hats, rubbers, and umbrellas should be provided. There should be an assortment of up-to-date magazines and light reading material, preferably in leather-bound covers to keep them from becoming prematurely dog-eared. The reading light should be adequate. Some physicians put up bulletin boards presenting information on health, education, and other interesting subjects. Others place writing desks in the reception room for patients' convenience or arrange a children's corner with small furniture and children's toys. Some physicians have found television helpful in keeping children occupied. Pictures, posters, plants, or cut flowers lend added charm to a reception room. A lighted aquarium or piped-in restful music may provide that extra touch that keeps patients loyal to their doctor.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Expert Witnesses: Cross-Examination Relating to Fee Charged for Testifying.—This was an action for damages for injuries sustained by a passenger in a bus-truck collision. From an unsatisfactory judgment for the plaintiff in the trial court, the plaintiff appealed to the superior court of Pennsylvania.

The plaintiff recovered a verdict in the trial court, but it was too small. He appealed, contending that the meager verdict was the result of restrictions that the trial court placed on the cross-examination of one of the defendant expert medical witnesses.

Under cross-examination of the physician, the plaintiff developed the fact that the physician had testified in many cases over a long period of time, for the defendant company and others, although he skillfully evaded exact numbers, which he called "statistics." The physician testified that he had received $50 for his pretrial examination of the plaintiff, and to the question, "What is your arrangement to be paid for testifying here?" he answered, "There are no arrangements made." He was then asked "How much do you expect to get paid for testifying here today?" The trial court sustained an objection to this testimony.

In a prior case the Supreme Court of Pennsylvania had said that it was pertinent for the jury to know what arrangements the doctors had made or were contemplating making for compensation for their services in the case and that the trial judge's refusal to require them to testify under oath and subject to cross-examination constituted a substantial error that made necessary the granting of a new trial. In the present case, to justify his ruling, the trial judge said: "A contemplated mutual arrangement and an *ex parte* expectation are very different things, for a man may expect more than he gets. If it be argued that the word 'expect' may be interpreted either as a mere hope or as reliance upon something certain but still in the future, it is our view that the latter meaning was eliminated by the doctor's statement that *no* arrangement had been made." On appeal, the judge said that apparently, under the opinion of the trial court, if the counsel had inquired concerning a contemplated arrangement instead of an expected arrangement his question would have been free from objection. The difference between the two words, said the court, is minute and vague. Both words describe "subjective mental processes"; both connote the mental state of looking forward; contemplation, as here employed, signifies a purpose in respect to the future; expectation is more than a bare hope, implying a high degree of certainty that a future event will occur. The prior Supreme Court decision, which used the expression "arrangements the doctors had made or were contemplating" clearly permits exploration of the future as well as of the past, of recompense that will be claimed as well as an express pretrial agreement to pay a stipulated fee. Continuing, the court said, however, that the logic of words must yield to the logic of realities. In this case a physician with 32 years of experience as a court witness was testifying. His experience and prestige have undoubtedly created a demand for his services that he is able to sell in a seller's market. In all probability he is not obliged to negotiate for fees; he commands them; and serves only those who meet his terms. It would be in keeping with the practice of other professional witnesses if he indeed fixed his fees unilaterally and ex parte. Nevertheless, the witness, drawing upon his experience and his practice in charging and receiving fees, could have stated an estimate of the monetary value he placed upon his services; that is to say, what he expected to charge and receive and, even if he could not, the question itself was not outside of the "reasonable limits" of interrogation. The amount of an expert's fees, whether stipulated in advance of a trial or determinable in the future, has a direct and vital bearing upon his credibility, his interest, bias, or partisanship. Accordingly the judgment was reversed and a new trial awarded. *Reed v. Philadelphia Transportation Company, 90 A. (2d) 371 (Pennsylvania, 1952).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Internal Medicine, Chicago

93:1-166 (Jan.) 1954

Clinical Usefulness of Determination of Circulating 17-Hydroxycorticosteroid Levels. G. T. Perkoff, A. A. Sandburg, D. H. Nelson and F. H. Tyler.—p. 1.
Function of Pancreatic Juice and of Bile in Assimilation of Dietary Triglyceride: Review. J. H. Annegers.—p. 9.
*Efficacy and Toxicity of Oxytetracycline (Terramycin) and Chlortetracycline (Aureomycin): With Special Reference to Use of Doses of 250 Mg. Every Four to Six Hours and to Occurrence of Staphylococcic Diarrhea. M. Finland, M. E. Grigsby and T. H. Haight.—p. 23.
*Infections Occurring During Antimicrobial Therapy. J. W. Hofer and G. M. McCaskey.—p. 44.
Successful Treatment of Intestinal Moniliasis with Fatty Acid-Resin Complex. I. Neuhauser.—p. 53.
*Use of Artificial Kidney in Chronic Renal Disease. F. Goldner, G. L. Gordon and L. E. Danzig.—p. 61.
Alcaptonuria and Ochronosis: Report of Five Cases Occurring in an American Family. R. L. Black, J. F. Lowney and P. M. Duffy.—p. 75.
Alpha Cells of Pancreas—Morphologic and Physiologic Considerations: Review. B. W. Volk, S. S. Lazarus and M. G. Goldner.—p. 87.
The Two-Component Mucous Barrier: Its Activity in Protecting Gastroduodenal Mucosa Against Peptic Ulceration. F. Hollander.—p. 107.

Oxytetracycline and Chlortetracycline.—An analysis is presented of the clinical results obtained in 520 patients treated with either oxytetracycline (Terramycin) or chlortetracycline (Aureomycin) for a large variety of diseases, but chiefly for infections of the respiratory or urinary tract. Most of the patients were given oral doses of 250 mg. every four hours or every six hours, but 37 of them received 500 mg. doses at similar intervals. The incidence of toxic effects accompanying the different dosage regimens was noted, and special attention was paid to the occurrence of staphylococcic diarrhea. Favorable clinical and bacteriological results were obtained with each of the dosage regimens in patients with acute infections due to susceptible types of organisms. The results with each of the two antibiotics were, on the whole, fairly comparable, considering the relatively small number but wide variety of diseases treated and the number and character of the underlying complicating factors. No conclusions could be drawn with respect to the relative efficacy of the dosage regimens employed as compared with the larger doses that had been used previously. The most frequent toxic effects were those involving the gastrointestinal tract. The frequency of the occurrence of gastrointestinal complications, particularly of severe watery diarrhea, was significantly greater in the patients who were treated with oxytetracycline than in those who received chlortetracycline in the same dosage. With each of these antibiotics diarrhea was about twice as frequent among patients who received 250 mg. doses every four hours or 500 mg. doses every four or six hours than among those given 250 mg. every six hours. Fecal cultures were made for 38 of the patients in whom diarrhea developed during oxytetracycline therapy and for 22 of those in whom it developed during chlortetracycline therapy. Hemolytic coagulase-positive strains of Micrococcus (Staphylococcus) aureus were found as the only or predominant organism in the fecal cultures of 27 of the former and 4 of the latter. Diarrhea subsided fairly rapidly, and the normal flora returned in the stools in most of the patients when the oral administration of the offending antibiotic was discontinued, and only general supportive measures were instituted promptly after the diarrhea began; this improvement occurred without relation to the subsequent use of other specific antibacterial agents or to the susceptibility of the fecal staphylococci to those agents.

Infections During Antimicrobial Therapy.—The authors present six case histories of patients in whom secondary infections developed during treatment with various antibiotics for primary infections. In each patient the relapse was caused by a microorganism that was resistant to the particular drug used. Superinfections of this kind occur in a small but significant number of persons receiving antimicrobial therapy. There are three basic types of mechanisms by which superinfection can result. 1. The drug may so alter the natural bacterial flora of the body that insensitive microbes become numerically predominant. If these initially scarce micro-organisms have the ability to invade the blood stream or specific tissues, a new infection may result. 2. The drug may actually stimulate bacterial growth, according to the Arndt-Schulz law that poisons in small doses are stimulants. This phenomenon has been demonstrated in vitro with penicillin. Garrod has shown that there are appropriate stimulating concentrations of penicillin for both sensitive and resistant species of bacteria. 3. The drug may bring about an alteration of certain tissues, as in clinical vitamin deficiency states, diarrhea with enterocolitis, and, reportedly, pseudomembranous enterocolitis.

Artificial Kidney in Chronic Renal Disease.—The artificial kidney is an important auxiliary in the treatment of chronic renal insufficiency. The four main indications for its use are (*a*) to obtain prolonged remissions in selected symptomatic patients, (*b*) to prepare patients for indicated surgical procedures, (*c*) to maintain patients in whom the delicate state of equilibrium in renal function is temporarily upset, and (*d*) to delay the progress of physiological abnormalities and the advent of more pronounced symptoms in relatively asymptomatic patients; more experience is needed to settle this last point. The factors that appear to predispose to a prolonged remission are (*a*) a general symptomatologic and chemical "plateau"; (*b*) the absence of severe generalized vascular disease, even though hypertension is present; (*c*) the absence of overwhelming infection or septicemia; (*d*) polyuria, and (*e*) a predialysis serum sodium level above 130 mEq. per liter. Favorable chemical responses were noted after 20 dialyses in 15 patients who had far advanced renal insufficiency of varying cause. After all 18 dialyses in which evaluation was feasible, the symptomatologic response was favorable. Short remissions occurred after 13 dialyses and long-term ones after 7 dialyses. The average duration of the short remissions was slightly longer than 3 days; the average duration of life after the 13 dialyses was 21 days. The longest of the long-term remissions was 104 days. One of these remissions lasted only 20 days, at which time death caused by myocardial infarction and peritonitis occurred; at the time of death symptoms associated with uremia had not reappeared. Although many patients with chronic renal insufficiency have been treated with the artificial kidney, more must be observed before any definite conclusions can be reached. It is emphasized that the series presented consisted mostly of patients in the terminal stages of their disease.

Alabama State Medical Assn. Journal, Montgomery

23:143-166 (Dec.) 1953

Gastroscopy. A. M. Freeman.—p. 143.
Gastric Resection for Complicated Peptic Ulcer. J. W. Donald and J. G. Donald.—p. 146.
Problem of Headache: Approach to Classification and Treatment. W. G. Haynes.—p. 150.

Periodicals on file in the Library of the American Medical Association may be borrowed by members of the Association or its student organization and by individual subscribers, provided they reside in continental United States or Canada. Requests for periodicals should be addressed "Library, American Medical Association." Periodical files cover only the last 11 years, and no photoduplication services are available. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each item. Only three periodicals may be borrowed at one time, and they must not be kept longer than five days. Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted.

American Journal of Clinical Pathology, Baltimore

23:1187-1296 (Dec.) 1953

Liver Lesions Following Intravenous Administration of Polyvinyl Pyrrolidone (PVP). E. A. Gall, W. A. Altemeier, L. Schiff and others.—p. 1187.

Benign Acquired Toxoplasmosis with Subsequent Pregnancy. M. F. Stanton and H. Pinkerton.—p. 1199.

Allergic Granulomatosis Associated with Visceral Larva Migrans: Case Report with Autopsy Findings of Toxocara Infection in Child. R. Brill, J. Churg and P. C. Beaver.—p. 1208.

Hypersensitivity to Bee Sting: Report of Fatal Case and Review of Literature. J. R. Schenken, J. Tamisiea and F. D. Winter.—p. 1216.

Anti-Jk^a: Three New Examples of Isoantibody: Frequency of Factor in Caucasians, Negroes and Chinese of New York City. R. E. Rosenfield, P. Vogel, N. Gibbel and others.—p. 1222.

Effect of Ethylenediamine Tetraacetic Acid on Cholesterol Metabolism in Rabbits: Preliminary Report on Effect of Parenteral and Oral Administration of Disodium and Calcium Salts. H. S. M. Uhl, H. H. Brown, A. Zlatkis and others.—p. 1226.

Fatal Gastroenteritis Associated with Clostridium Welchii. E. Kellert and W. Meeler.—p. 1234.

Mixed Tumor of Parotid Gland with Tyrosine Crystals in Matrix: Report of Case. W. K. Bullock.—p. 1238.

Pathology of Anomalies of Thoracic Aorta. J. E. Edwards.—p. 1240.

American Journal of Medical Sciences, Philadelphia

226:597-718 (Dec.) 1953

Use of Correctives in Prevention of Barbiturate Intoxication. T. Koppanyi and J. F. Fazekas.—p. 597.

Survival Rates After Myocardial Infarctions With and Without Long Term Anticoagulant Therapy. J. W. Keyes, E. H. Drake, T. N. James and F. J. Smith.—p. 607.

Stingray Injuries: Review and Discussion of Their Treatment. F. E. Russell.—p. 611.

*1-Hydrazinophthalazine (Apresoline) in Treatment of Hypertensive Disease: Clinical Trial with Control Group. D. H. Merrill and K. Kenyon.—p. 623.

Modification of Hypotensive Action of 1-Hydrazinophthalazine (Apresoline) by Priscoline, Hexamethonium and Section of Carotid Sinus and Aortic Buffer Nerves in Normal and Renal Hypertensive Dogs. J. W. McCubbin and I. H. Page.—p. 632.

*Evaluation of Treatment in Hypertension: Effects of Cinchona Alkaloids. A. W. Seligmann, F. C. Ferguson Jr., S. Garb and others.—p. 636.

On the Relationship of Urinary Copper Excretion to Aminoaciduria in Wilson's Disease (Hepatolenticular Degeneration). L. L. Uzman.—p. 645.

Effect of Norepinephrine and Epinephrine on Renal Hemodynamics. L. C. Mills, J. H. Moyer and J. M. Skelton.—p. 653.

Differentiation Between Congenital and Acquired Forms of Hemolytic Anemia: Observations on 47 Cases of Hereditary Spherocytosis and 24 Cases of Autoimmune Hemolytic Disease. L. E. Young and G. Miller.—p. 664.

Stress Urinary Incontinence in Female: Review of Modern Approach to This Problem. J. J. Mikuta and F. L. Payne.—p. 674.

Importance of Trypsin in Infancy and Childhood. P. W. Guilbert and I. J. Wolman.—p. 688.

Hydralazine (Apresoline) Hydrochloride in Treatment of Hypertension.—The results obtained by giving only hydralazine (Apresoline) hydrochloride to one group of ambulatory patients with hypertension were compared with those obtained by giving placebos of similar appearance to another group. The alternate patient method was used in determining the distribution of the drug and the placebo. All patients were seen by the same observer at each clinic visit, and all were apparently benefited by the regular visits to the same physician. Some lowering of the blood pressure was found in each group, but the decline in the patients receiving hydralazine was statistically more significant that that found in those given the placebos. Patients vary greatly in their response to hydralazine; of the 37 receiving the drug, 12 had a drop in diastolic pressure of 20% or more (either while supine or sitting), some showed no change, and in 2 the diastolic pressure increased. Side-effects, chief among which was an occipital headache, were common, and they were severe enough to necessitate discontinuance of the drug in 11 patients. Two of the 28 patients receiving placebos discontinued them because of headache and nausea that they felt were drug-induced. A drop of more than 20% in the diastolic pressure, while sitting, was found in only one of the patients in the placebo group. Comparison of the figures obtained in patients who have been receiving either hydralazine or the placebo for more than six months shows that the significant difference between the two groups is persistent, indicating that the drug is effective even after long-continued use. The response of the individual patient, however, cannot be predicted. Three hospitalized patients with nitrogen retention, who were among several treated with hydralazine but not included in the study groups, appeared to be adversely affected, suggesting that hydralazine should not be given to patients with uremia.

Effects of Cinchona Alkaloids.—The long-term effect of cinchona alkaloids on the blood pressure was studied by giving quinidine sulfate, quinine sulfate, and a lactose placebo, dispensed in identical capsules identifiable only by code number, to 44 patients with long-standing hypertension. The double blind principle was used throughout the study so that neither the patient nor the attending physician knew which substance was being given at any particular time. The patients were told that the test materials were "medicine for the blood pressure" and were instructed to take three capsules a day, one before each meal. Each capsule contained 0.3 gm. of a drug, and this dosage (0.9 gm. per day) was considered to be the maximum amount of alkaloids that could be taken regularly by an unselected group of patients. Undesirable side-effects were common even with this dosage, and many patients were unable to tolerate it, especially when quinine was being given. The patients, divided into two random groups at the first visit, received courses lasting four weeks of quinidine sulfate and placebo in alternate order. At least two weeks of observation without therapy followed, and then the patients, redivided into two groups at random, were given quinine sulfate and placebo alternately in courses lasting four weeks, as before. Statistical analyses alone do not permit accurate evaluation of drug therapy in hypertension, because treatment in itself frequently produces a marked reduction in the blood pressure even of stabilized hypertensive patients. The double blind method, however, was apparently successful in controlling the nonspecific effects of therapy. Summaries of the data obtained, prepared by one who had no contact with the patients, showed that the beneficial effects apparently produced by the cinchona alkaloids on the blood pressure and symptoms of these patients were equalled in each case by those of the lactose placebo. Subjective factors proved unreliable as guides to the result of treatment; both the patients and the attending physicians expressed opinions at variance with the condition of the pressure and with each other. Hypertensive symptoms, in fact, seem to have little, if any, connection with hypertension itself.

Am. Practitioner & Digest of Treatment, Philadelphia

4:813-900 (Dec.) 1953

Some Clinical Aspects of Disturbed Pituitary Function. E. P. McCullagh.—p. 813.

Use of Mood Ameliorating Agents in Treatment of Psychogenic Obesity. P. Rosenberg.—p. 818.

Diagnostic Problems of Simple Gastroduodenal Ulcer. H. B. Eisenstadt and B. B. Elster.—p. 821.

Treatment of Angina Pectoris, Including Angina Decubitus and Status Anginosus. H. L. Jaffe.—p. 828.

Diagnosis and Management of Lesions of Vulva. H. Packer.—p. 834.

Cardiac Emergency Kit: Recommended Plan for Hospital Use. J. J. Silverman and M. Trudeau.—p. 840.

Life Stress and Gastro-Intestinal Diseases. W. J. Grace.—p. 848.

Polycythemia Vera. C. F. Stroebel.—p. 853.

Significance of Size and Radiographic Density of Solitary Lesions in Lungs. J. R. Vivas and S. F. Crabtree.—p. 857.

*Fibrous Adhesive Pericarditis Following ACTH Therapy in Acute Rheumatic Pancarditis: Case Report. R. F. Sheely.—p. 862.

Fibrous Adhesive Pericarditis Following Corticotropin Therapy for Rheumatic Pancarditis.—Sheely presents the case of a 10-year-old girl in whom acute rheumatic pancarditis developed after an attack of acute tonsillitis. Treatment with adequate amounts of corticotropin was followed by the development of an intractable congestive state characterized by hepatomegaly, recurrent tense ascites, and dependent edema. This congestive state typical of Pick's syndrome occurred approximately two months after institution of therapy with 800 mg. of corticotropin, at a time the patient was clinically improved, with great improvement in appetite, sense of well-being, and subsidence of toxicity. At no time during the 17 months of hospitalization was fluid in the pleural spaces or persistent clinical signs of pulmonary congestion detected in repeated physical examinations and roentgenographic studies. In the

absence of a presystolic murmur, it was the clinical impression that the patient had constricting pericarditis with resultant enlargement of the right side of the heart and portal hypertension. Necropsy revealed a dense fibrous adhesive pericarditis; there was no true mitral stenosis. This condition produced the clinical picture of a constrictive pericarditis. Although the use of corticotropin cannot be definitely linked to the pathological lesions in this case, the occurrence of Pick's syndrome two months after steroid therapy leads to interesting considerations in the light of recent observations on the effect of these steroids in other vascular conditions. Shick and others recently observed, in pathologically proved cases of periarteritis nodosa treated with corticotropin and cortisone, that massive fibrosis resulted in the involved arterioles and medium-sized arteries extensive enough to obliterate these vessels, producing infarctions in the involved visceral organs.

Bacteriological Reviews, Baltimore

17:249-338 (Dec.) 1953

Microbiologists in Transition. G. M. Dack.—p. 249.
Use of Metabolically Blocked Organisms for Analysis for Biosynthetic Pathways. E. A. Adelberg.—p. 253.
Lysogeny. A. Lwoff.—p. 269.

17:339-448 (Dec. [Supplement]) 1953

Immunization Against Poliomyelitis. T. E. Boyd.—p. 339.

Blood, New York

8:1061-1156 (Dec.) 1953

Optical Properties of Hemoglobin in Microdrepanocytic Disease. A. Ascenzi and E. Silvestroni.—p. 1061.
*Hyperglobulinemia as Cause of Hemophilia-Like Disease. I. M. Nilsson and A. Wenckert.—p. 1067.
Factor in Lungs of Anaphylactically Shocked Guinea Pigs Which Can Induce Eosinophilia in Normal Animals. M. Samter, M. A. Kofoed and W. Peper.—p. 1078.
Plasma Factor for Platelet Adhesiveness and Clot Retraction Acceleration. J. P. Savitsky.—p. 1091.
Drowning Hemoglobinuria. C. E. Rath.—p. 1099.
Blood Volume and Molybdenum Toxicity in Rabbits. J. D. Burke, L. R. Arrington and G. K. Davis.—p. 1105.
Finding of L. E. (Lupus Erythematosus) Cells in Smears of Untreated, Freshly Drawn Peripheral Blood. B. Chomet, M. M. Kirshen, G. Schaefer and P. Mudrik.—p. 1107.
Red Blood Cell Survival Studies. G. S. Eadie and I. W. Brown Jr. —p. 1110.
Case of Erythremic Myelosis (Di Guglielmo). J. Báez-Villasenor and L. Sánchez-Medal.—p. 1137.

Hyperglobulinemia as Cause of Hemophilia-Like Disease.—Nilsson and Wenckert of Malmö, Sweden, call attention to the fact that in recent years several cases of a formerly unrecognized coagulation defect have been described. This defect is characterized by a hemophilia-like hemorrhagic state and caused by the presence in blood of a circulating anticoagulant that is active in the first phase of coagulation and that is neither heparin nor anticephalin. The first case was reported in 1940 by Lozner, Joliffe, and Taylor. Thereafter, more than 20 similar cases have been described. In 1950 the term "Hemmkörper" (inhibiting body) hemophilia was applied by an Austrian investigator. Other workers reported the presence of circulating anticoagulants antagonistic to thromboplastin or to the formation of thromboplastin in patients with true hemophilia following multiple transfusions. No clear-cut distinction can be made between these two groups. The first group comprises only genuine hemophiliacs, that is, only men, while the second includes both men and women. The authors present a case of probably familial hyperglobulinemia. A 44-year-old woman had a circulating anticogulant, antagonistic to the initial phase of the coagulation, most likely to the antihemophilic globulin. The anticoagulant factor was separated by means of salt precipitation and by electrophoresis. It was shown to be identical with one of the patient's abnormal gamma globulin. There is no reason to suppose an isoimmunization process. The coagulation defect is, therefore, assumed to be a secondary phenomenon to the patient's probably familial protein disturbance. Dysproteinemia has been observed in hemophilia-like diseases several times, but the etiological connection between the coagulation anomaly and the protein disease has not been previously established. On the other hand, even serious dysproteinemias seem rarely to be complicated by a circulating anticoagulant. A connection between the sedimentation rate and the coagulation of the blood was shown to be present in this patient.

Circulation, New York

8:801-960 (Dec.) 1953

Electrocardiographic Pattern of Hypopotassemia With and Without Hypocalcemia. B. Surawicz and E. Lepeschkin.—p. 801.
*Studies on Control of Hypertension by Hyphex: II. Toxic Reactions and Side Effects. J. D. Morrow, H. A. Schroeder and H. M. Perry Jr. —p. 829.
Newer Drugs in Treatment of Hypertension: I. Use of Hexamethonium Salts. H. A. Sieber, K. S. Grimson and E. S. Orgain.—p. 840.
Problems in Diagnosis and Surgical Treatment of Pulmonic Stenosis with Intact Ventricular Septum. J. W. Kirklin, D. C. Connolly, F. H. Ellis Jr. and others.—p. 849.
Orthostatic Factors in Pulsus Alternans. B. Friedman, W. M. Daily and R. S. Sheffield.—p. 864.
Effects of Papaverine upon Ectopic Ventricular Tachycardia Produced by Myocardial Infarction. A. S. Harris, A. Estandía, A. Bisteni and H. T. Smith.—p. 874.
Evaluation of Routine Serial Fluoroscopic Examinations of Heart in Postero-Anterior and Oblique Views at Specific Degrees of Rotation, with Special Reference to Angle of Clearance of Left Ventricle. M. G. Wilson, N. Epstein, H. N. Helper and K. Hain.—p. 879.
*Heart Force Effects of Sympathomimetic Amines as Basis for Their Use in Shock Accompanying Myocardial Infarction. P. C. Gazes, L. I. Goldberg and T. D. Darby.—p. 883.
Respiratory and Circulatory Studies of Patients with Mitral Stenosis. P. C. Curti, G. Cohen, B. Castleman and others.—p. 893.
Further Studies in High Fidelity Electrocardiography: Myocardial Infarction. P. H. Langner Jr.—p. 905.
Some Requirements in Equipment and Technics for Vectorcardiography. J. A. Cronvich, G. E. Burch and J. A. Abildskov.—p. 914.
Normal Electrocardiogram. C. E. Kossmann.—p. 920.

Control of Hypertension by Hyphex.—Hyphex, a mode of therapy in which hydralazine (Apresoline) is combined with hexamethonium chloride, was used for 6 to 25 months in the treatment of 258 patients with hypertension. Untoward reactions are reported. Minor unwanted effects of hexamethonium chloride, which produces a partial blockade of all autonomic ganglions consisted of orthostatic hypotension and suppression of intestinal motility with resulting constipation; blurring of vision, which occurred frequently; dryness of mouth and hypotonicity of the bladder, which were common; decreased potency or impotence in men, which was constant in the early stage but complete or partial recovery was noted in many; and cold intolerance, which was frequently observed. Minor reactions to hydralazine consisted of stuffy nose, periorbital edema, lacrimation, headache, generalized aching, and prostration in most of the patients on initiation of therapy, but these varied greatly in severity. All of these symptoms were milder and of shorter duration when hydralazine was used in combination with hexamethonium chloride, than when hydralazine was used alone. Fatal complications were observed in the form of interstitial pneumonia in five patients, uncontrolled hypotension in one, and sudden death in one. Hydralazine apparently caused arthritis and "collagen-like" disease in 16 patients and repeated febrile responses in 6. Serious obstructive phenomena caused by hexamethonium chloride in previous organic obstruction occurred in 10 patients. Of the 44 patients (17%) with untoward reactions, 8 (3%) died, 5 of interstitial pneumonia, 1 of pneumonia after parotitis, and 2 from simultaneous administration of both drugs. Despite untoward reactions and undesirable side-effects, the combined use of hydralazine and hexamethonium chloride is an effective and practical method of treating severe hypertension provided precautions are taken to minimize serious consequences, for the benefits observed clearly have outweighed the risks. Neither agent is ideal.

Treatment of Shock Accompanying Myocardial Infarction.—Of 14 patients who were between the ages of 39 and 70 and who were in severe shock accompanying myocardial infarction, 7 were treated with intravenous infusions of phenylephrine (Neo-Synephrine) hydrochloride and the others received intravenous infusions of levarterenol (Levophed). The latter was administered in a solution containing 4 mg. of levarterenol and 5% dextrose in each liter of distilled water. Phenyl-

ephrine was administered in a concentration of 10 mg. per liter in two patients and 20 mg. per liter in the others. The rate of infusion flow was determined by the level of systolic blood pressure that was maintained below 120 mm. Hg in each patient except one, a known hypertensive with a blood pressure of 134/90. The rate of infusion flow ranged from 5 to 100 drops per minute. If the blood pressure remained stable with the slower flow rate, the pressor amine administration was discontinued. If shock recurred, the infusion was restarted. Twelve of the 14 patients were in congestive heart failure. Six patients in congestive heart failure treated with levarterenol recovered. Five patients in congestive heart failure treated with phenylephrine responded with an immediate rise of blood pressure, but the pressure failed to stabilize and the patients succumbed despite continuous infusions of phenylephrine. Two patients with no evidence of congestive heart failure were brought out of shock by phenylephrine. One of these patients recovered; the other died 48 hours after the phenylephrine infusion was discontinued. One patient treated with both sympathomimetic amines failed to respond to phenylephrine and was later brought out of shock by levarterenol. This patient died 18 hours after discontinuance of levarterenol therapy. Determinations of relative contractile force and arterial pressure changes were carried out after 51 intravenous injections of *l*-epinephrine, levarterenol and phenylephrine in unanesthetized dogs. Results showed, that levarterenol produced pronounced increments in heart contractile force, in addition to pressor effects in the experimental animals. The contractile force increments were of similar magnitude to those produced by *l*-epinephrine. Phenylephrine was observed to be more predominantly a pressor amine, producing only minimal changes in heart contractile force. The effect on contractile force of the heart of levarterenol is presented as a basis for the high recovery rate observed with this amine in the treatment of shock accompanying myocardial infarction, particularly in patients with associated congestive heart failure.

Delaware State Medical Journal, Wilmington

25:309-330 (Nov.) 1953

Present Status of Management of Urinary Infections. T. R. Fetter. —p. 309.
*Scrotal Diverticulum of Urinary Bladder. B. S. Vallett.—p. 316.
Intestinal Obstruction. G. S. Serino.—p. 318.
Rational Use of Hormones in Pregnancy. R. C. Hayden.—p. 320.
Alcoholism and General Practitioner. H. T. McGuire.—p. 322.
New Psychiatric Diagnostic Nomenclature. P. J. Polnsard.—p. 324.
Psychotherapy. W. A. Byrne.—p. 325.
The Physician's Role in Marital Problems. C. J. Katz.—p. 326.

25:331-364 (Dec.) 1953

Diplopia with Intracranial Aneurysm. J. R. Fox and E. R. McNinch. —p. 331.
Mycotic Rat-Bite Fever: Report of Case. D. A. Toren.—p. 334.
Clinicopathologic Case Report. J. Elliott, J. W. Annand and O. J. Pollak. —p. 335.

Scrotal Diverticulum of Urinary Bladder.—The case presented by Vallett was that of a man, aged 68, who was hospitalized with the chief complaint of not being able to void. He had difficulty in passing urine for 10 years. There was a doughy mass in the right scrotal sac. Rectal examination revealed benign overgrowth of the prostate. An excretory urogram showed prompt appearance of the dye in both kidneys. A 60 minute film showed the bladder incompletely filled and irregular in outline on its right lateral aspect. This suggested some factor responsible for drawing the bladder to the right. Tumor was thought of, but in a lesion of this extent there would have been a history of hematuria. Both ureters in their lower one-third were displaced to the right. Cystoscopy revealed a normal bladder mucosa, but the mucosal surface of that part of the bladder wall that appeared displaced to the right in the excretory films could not be seen. It was assumed that this was a local sacculated area consequent to bladder neck obstruction. A diagnosis of benign overgrowth of prostate and right hydrocele was made and an operation planned to resect the hydrocele, and this was to be followed by electric endoscopic vesical neck resection. The right scrotal sac was opened, and it became apparent that there was no hydrocele. A mass was seen that was doughy and compressible. Careful dissection of the mass revealed bladder muscle, and an incision into the lumen showed a bladder diverticulum. There was no urine in the diverticulum. A finger could be passed through the neck of this structure into the bladder proper. A transverse suprapubic incision was made and the bladder opened. It was now possible to invert the diverticulum and push it up through the external ring on into the bladder proper. However, it was apparent that diverticulectomy could be carried out more easily from the scrotal area. Consequently, the diverticulum was returned to this area and resected, purse-stringing its neck, which in turn was pulled up through the external inguinal ring, inverted, and tied from within the bladder. The prostate was left for subsequent endoscopic vesical neck resection that was carried out 18 days later. Diverticulum of the bladder in the scrotum is rare. The author makes a plea for the routine employment of the cystogram in urologic examination. The information gained is often greater than that derived from cystoscopy. It is the only accurate method of demonstrating scrotal cystocele.

Diseases of Chest, Chicago

24:591-700 (Dec.) 1953

*Clinical Observations on Viomycin Sulfate in Treatment of Tuberculosis. R. L. Hackney, E. Q. King, E. E. Marshall and others.—p. 591.
Dramamine (Dimenhydrinate) as Adjunct to PAS (Para-Aminosalicylic Acid) in Treatment of Pulmonary Tuberculosis. L. I. Leonard.—p. 601.
Stress in Pulmonary Tuberculosis: I. Thorn Test and Circulating Eosinophils in Surgical Patients. A. Goldman, I. A. Breckler, E. Stern and R. Robinson.—p. 608.
Continuous Pneumo Apparatus: Hospital and Portable Models. J. W. Cutler.—p. 619.
*Causes of Death in Carcinoma of Lung in Large Public Hospital: Analysis of 186 Fatalities. F. F. Boyce.—p. 626.
Improved Cardiac Defibrillator. R. M. Shepard Jr.—p. 642.
Congenital Multiple Cysts of Lung. H. Lichtenstein.—p. 646.
Experiences with Tetraethyl Ammonium Chloride in Bronchial Asthma. L. Gregory, M. Damiani, M. Johanson and others.—p. 655.
Experiences with Planography. B. Pollak.—p. 663.
Anterior Mediastinal Chondromyxosarcoma. A. Schwinger and S. D. Hemley.—p. 670.
Byssinosis. A. G. Hollander.—p. 674.
Tuberculous Stenosis of Trachea: Report of Case Treated by Incision. D. M. MacRae, J. J. Quinlan, F. J. Misener and J. E. Hiltz.—p. 679.
Audio-Visual Educational Methods in Cardio-Pulmonary Disease. J. S. Butterworth.—p. 684.

Viomycin Sulfate in Tuberculosis.—Five patients with advanced pulmonary tuberculosis and without history of previous antimicrobial therapy were treated with 3 gm. of viomycin sulfate daily for 13 weeks. The drug had a definite antimicrobial effect, but was too toxic for widespread use in tuberculosis therapy. Thirty-five patients with pulmonary tuberculosis were given 2 gm. of viomycin sulfate twice weekly and 12 gm. of *p*-aminosalicylic acid daily, and 24 control patients with pulmonary tuberculosis were given 2 gm. of dihydrostreptomycin twice weekly and 12 gm. of *p*-aminosalicylic acid daily. Viomycin sulfate in doses of 2 gm. twice weekly combined with 12 gm. of *p*-aminosalicylic acid daily was of slight toxicity. Tinnitus may be expected to occur. Allergic symptoms were not a major problem. Auditory nerve damage of a serious degree was not frequent. Streptomycin-resistant organisms were affected by viomycin, and patients having such organisms improved under intermittent viomycin treatment. Bacterial resistance to viomycin did not develop to a significant degree among patients treated for six months. Clinical improvement and clearing of exudative lesions evident roentgenologically were more striking in the patients treated with dihydrostreptomycin combined with *p*-aminosalicylic acid; however, the effectiveness of viomycin combined with *p*-aminosalicylic acid was definite and demonstrable. The clinical use of viomycin is feasible in the presence of streptomycin-resistant organisms and allergic sensitivity to streptomycin. Its use with newer antimicrobials should be explored.

Death from Carcinoma of the Lung.—Boyce analyzes 186 fatal cases of carcinoma of the lung from the Charity Hospital of Louisiana at New Orleans. Of the 186 patients, 33 underwent pneumonectomy and 22 of these died during the immediate postoperative period. Eleven others, who survived the operation, died later in the hospital. Only 4 of the 11 lived for two years or more, and only 1 lived as long as five years. Twenty-six of the 33 fatal pneumonectomies were palliative; this means

that the risk was greater in them than it would have been in curative operations. In 20 nonsurgical cases, in which the duration of symptoms before medical consultation was a month or less, 2 patients lived for a year after they became clinically ill, but 2 others lived for only two weeks, 3 for less than a month, and 5 for less than two months. These fatalities illustrate the unhappy and paradoxical fact that the shorter the duration of symptoms, the shorter also is the duration of life after their onset. The correlation between brevity of symptoms and the total illness was also clear in many of the 23 patients who submitted to surgical exploration, but the duration of life in this group was slightly longer than in the nonsurgical group. The extraordinarily wide extent and rapid development of extension and metastases in carcinoma of the lung was illustrated by 21 cases of metastases to the liver, 18 to the brain, 16 to the bones, 13 each to the adrenal glands and the heart and pericardium, 12 to the cervical lymph nodes, 11 to the aorta, 7 to the superior vena cava, 6 to the supraclavicular lymph nodes, 4 to the kidneys, 3 to the pancreas, and 2 each to the intestines and the thyroid. Only when pneumonectomy can be carried out promptly in respect to pathological process, not symptoms, are the results of treatment rewarding, but few patients were operated on promptly because they often delayed seeking treatment. Physicians whom they first consulted and hospital staff members often failed to suspect the diagnosis or were slow to recommend surgery. The investigation was sometimes perfunctory, sometimes unnecessarily repetitious, and sometimes enmeshed in hospital routine and protocol. Appallingly often the clinical course was far too brief to permit any positive action. If the mortality is to be lowered in carcinoma of the lung, which is now of major importance among malignant diseases, the profession must (1) develop a far higher index of suspicion concerning it; (2) explore the chest on suspicion much oftener; (3) stress the urgency of diagnosis and treatment; (4) investigate the whole person rather than limited fields whenever adults seek medical care for any reason whatsoever; and (5) make the fullest possible use of data available in mass chest surveys.

Geriatrics, Minneapolis

8:629-686 (Dec.) 1953

Preparation of Aged Patient Facing Emergency Surgery. S. E. Ziffren and R. F. Sheets.—p. 629.
Effect of Hydrogenated Alkaloids of Ergot on Hypertensive Headaches. T. Winsor.—p. 636.
Physical Medicine in Treatment and Rehabilitation of Rheumatic Disease. H. Dinken.—p. 643.
Use of Betaine-Lipotropic Combinations in Clinical Practice. L. M. Morrison.—p. 649.
Study of Socialization of Old People. W. Donahue, W. W. Hunter and D. Coons.—p. 656.

Industrial Medicine and Surgery, Chicago

22:547-602 (Dec.) 1953

Fatigue—Measurement and Reduction. L. Brouha.—p. 547.
Hydrocortisone Therapy in Ganglia. W. F. Becker.—p. 555.
Disability, Rehabilitation and Law of Damages: Implications of Modern Rehabilitation Medicine. F. A. Hellebrandt.—p. 558.
Cadmium Intoxication: Clinical Report of Acute Accidental Group Exposure. M. L. Amdur and R. A. Caputi.—p. 561.
*The Painful Elbow. G. G. Maida.—p. 567.
Radiation Injury and Present Status of Therapeutics: Bibliographic Review. T. J. Haley.—p. 569.
Lead and Lead Poisoning in Early America—The Pewter Era. C. P. McCord.—p. 573.
Prevention of Cardiac Disability. L. H. Bronstein, H. J. Bennett, S. C. Franco and others.—p. 578.

Painful Elbow.—An insidious condition of the elbow, which is found with relative frequency in heavy industry, is epicondylitis. This is a periostitis of the bone, affecting either the median or lateral epicondyle of the humerus, usually with some concomitant involvement of the nerves surrounding the area—the median, the radial, and/or the ulnar. The radiohumeral ligaments and even the bursas may be involved. It is the result of repeated bruising of the elbow. It causes moderate pain, and at the beginning even exquisite tenderness. Motion of the arm and forearm may be limited, but it is not definitely restricted. The pain shoots up and down the arm and forearm from the lateral or median epicondyle and usually is intensified by grasping or lifting with the extended forearm. The condition is chronic and largely refractory to treatment, but with the passage of time, it regresses spontaneously. During the last two years, the author diagnosed 20 cases, 3 of which are presented. Numerous treatments all proved to be of temporary value or just a means of tiding over during the acute stage. There is loss of efficiency but no disability. The Workmen's Compensation Commission's final opinion was always discomfort over the elbow region, no disability.

International Journal of Leprosy, New Orleans

21:295-426 (July-Sept.) 1953

Leprosy in Children of French West Africa. Laviron and Lauret.—p. 295.
*BCG in Prophylaxis of Leprosy: Preliminary Report. N. de Souza Campos.—p. 307.
*Value of Lepromin Reaction in Diagnosis of Clinical Forms of Leprosy. S. Schujman.—p. 313.
Study of Morphological Modifications of Mycobacterium Leprae During Chemotherapy. M. G. Malfatti and E. D. L. Jonquieres.—p. 323.
Staining of Mycobacterium Leprae by Rio Hortega Silver Method in Frozen and Paraffin Sections. J. Sanchez.—p. 331.
Review of Recent Animal Inoculation Studies with Human and Murine Leprosy Bacilli. T. Tanimura and S. Nishimura.—p. 335.

BCG in Prophylaxis of Leprosy.—De Souza Campos orients his note toward three questions asked by Fernandez in 1951. 1. What is the value of the positive Mitsuda reactivity induced by BCG in a healthy person? 2. What is the incidence of leprosy among persons vaccinated with BCG and among those not so vaccinated? 3. Are there well-substantiated cases of lepromatous leprosy developing in persons previously vaccinated with BCG? From the small number of statistics that exist, the following conclusions can be drawn. BCG vaccination clearly has a protective effect against leprosy infection, as shown by the incidence of only 0.6% among the vaccinated persons as against 5.6% among the nonvaccinated ones during the same period of time. Among the vaccinated contacts, only tuberculoid leprosy developed, reactional in five, the childhood nodular form in four, and circinate in one. In contrast, of the cases among the nonvaccinated contacts 26.3% were of the lepromatous form, 46.9% of the undifferentiated form, and only 26.8% of the tuberculoid form. These data are obviously initial ones, and subsequent observations may alter them quantitatively and qualitatively. There is no doubt in the author's mind, however, that they indicate clearly that BCG has a protective effect against leprosy infection.

Lepromin Reaction in Diagnosis.—Although the international classification of leprosy is based on clinical, bacteriological, histological, and immunological criteria, the author feels that the immunological one as revealed by the late reaction to the lepromin test (Mitsuda phenomenon) is the most important and dependable one because it is the least subject to change. In the prolonged evolution of the disease, the clinical, bacteriological, and histological elements not only undergo modification but may eventually disappear entirely, whereas the immunological one remains, as a rule, constant, positive in the tuberculoid form and negative in the lepromatous form. With respect to the indeterminate group, the author has never seen a strongly positive case change to negative or evolve to the lepromatous form. On the other hand, cases with negative lepromin reactions, especially those that have positive bacteriological evidence and that are insufficiently treated, usually change to the lepromatous form. The number of cases observed is, however, relatively small, and, to permit definite conclusions to be drawn, indeterminate cases should be studied thoroughly at other centers and immunological curves should be established by testing every three months to demonstrate immunological evolution. If it is proved that the cases with negative lepromin reactions do eventually become the lepromatous form, they should be regarded as prelepromatous or potentially lepromatous. The lepromin reaction assumes its full value in classification in the cases without skin lesions, since guidance from clinical, bacteriological, or histological aspects is lacking. Where the Mitsuda reaction is frankly negative, it may be concluded that the disease is of the lepromatous form with nerve localization, if the patient has never had skin lesions or a neural form secondary to a previously lepromatous

one in which there were skin lesions that have disappeared. When the reaction is frankly positive the disease can generally be regarded as tuberculoid leprosy of the nerve where there have never been skin lesions or neural, secondary to previously tuberculoid leprosy, when skin lesions have disappeared. Two cases are reported to illustrate how the immunological findings alone permitted diagnosis of the clinical form; these diagnoses were confirmed by biopsy of the cubital nerve.

Journal of Bacteriology, Baltimore

66:627-738 (Dec.) 1953. Partial Index

External Surface of Human Body as Habitat for Nonfermenting Non-pigmented Yeasts. G. H. Connell and C. E. Skinner.—p. 627.
Desoxyribonucleic Acid and Budding Cycle in Yeasts. M. Ogur, S. Minckler and D. O. McClary.—p. 642.
Selective Action of Thallium Acetate and Crystal Violet for Pleuropneumonia-like Organisms of Human Origin. H. E. Morton and J. G. Lecce.—p. 646.
Influence of Diphosphopyridine Nucleotide on Stability of Typhus Rickettsiae. M. R. Bovarnick, E. G. Allen and G. Pagan.—p. 671.
Acid Formation in Cultures of Mycobacterium Butyricum. G. J. E. Hunter.—p. 676.
Metabolism of Ribonucleic Acid in Normal and Bacteriophage Infected Escherichia Coli. L. A. Manson.—p. 703.

Journal Clin. Endocrin. & Metab., Springfield, Ill.

13:1445-1604 (Dec.) 1953

17-Hydroxycorticosteroids and 17-Ketosteroids in Urine of Human Subjects; Clinical Application of Method Employing β-Glucuronidase Hydrolysis. A. A. Sandberg, D. H. Nelson, E. M. Glenn and others. —p. 1445.
Methods for Determination of Formaldehydogenic Steroids by Diffusion and Direct Reaction. H. Wilson.—p. 1465.
Primary Gonadal Insufficiency in Girl and Boy: Metabolic Effects of Estrogen and Testosterone. F. T. G. Prunty, R. R. McSwiney and B. E. Clayton.—p. 1480.
Effect of Ingested Protein and Tyrosine on Circulating Eosinophils. I. Vartiainen and J. Apajalahti.—p. 1502.
*Propylthiouracil: 4-6 Year Follow-Up of Selected Patients with Graves' Disease. E. P. McCullagh and C. B. Cassidy.—p. 1507.
*Histologic Lesions in Thyroid Glands of Patients Receiving Radioiodine for Hyperthyroidism. M. B. Dailey, S. Lindsay and B. R. Miller. —p. 1513.
Carcinoma of Thyroid Gland. J. D. Majarakis, D. P. Slaughter and W. H. Cole.—p. 1530.

Propylthiouracil in Graves' Disease.—Propylthiouracil was given to 60 patients with Graves' disease who had small to moderate-sized diffuse goiters and hyperthyroidism. All patients were followed from four to six years after treatment with propylthiouracil was discontinued. In 40 (66.7%) of the 60 patients thyrotoxicosis was in remission at least four years after the drug was discontinued. The remaining 20 patients (33.3%) had relapses, and in 13 of them a relapse occurred within five months after treatment was discontinued. Remission implies complete freedom from symptoms and signs of hyperthyroidism and a basal metabolic rate lower than + 10%. In 32 of the 40 patients (80%) who had lasting remissions, the hyperthyroidism was completely controlled within three months after treatment was started. In most patients, a dose of 200 mg. of propylthiouracil per day given as four 50 mg. tablets (one after each meal and one at bed time) was adequate for complete control of the hyperthyroidism. Because of occasional slow response, the amount of the drug was increased to 300 or 400 mg. per day if necessary. It is the authors' impression that the longer treatment is continued the more likely is the chance of lasting freedom from the thyrotoxicosis, but, in 8 of the 40 patients in whom the disease remained in remission, treatment was stopped five months or less after control was first obtained.

Histopathological Lesions in Thyroid After Radioiodine Therapy.—The hyperplastic or nodular thyroid glands of 23 hyperthyroid patients were irradiated with radioactive iodine (I^{131}) and examined microscopically. The patients treated toward the end of the study received 120 μc of I^{131} per estimated gram of thyroid tissue, while earlier in the study small doses of I^{131} were employed. The total dose of I^{131} administered varied from 471 to 23,439 μc. At the time of thyroidectomy, 21 patients were euthyroid and in most of them the thyroid gland had decreased in size. Of the remaining two patients, one died from diabetic acidosis and the other from laryngeal spasm during thiopental (Pentothal) sodium administration because of a manic state; both of these patients were still hyperthyroid. On microscopic examination thyroid glands of seven patients were predominantly hyperplastic and no definite radiation effects were observed. The thyroid glands of nine patients had lesions identical with those of Hashimoto thyroiditis. This high incidence of thyroiditis, when compared with that in nonirradiated hyperplastic thyroids, is highly signficant statistically. This suggests that Hashimoto thyroiditis is one effect of internal irradiation with radioiodine. Four thyroid glands contained multiple nodules and showed moderate follicular atrophy of the residual glandular tissue. It was concluded that this atrophy was also the result of injury by irradiation. Three thyroids showed diffuse follicular atrophy and fibrosis, regarded as classic lesions caused by irradiation. Vascular disease in the 23 thyroid glands was minimal and had no relation to the regressive thyroid lesions observed. Pleomorphic and hyperchromatic alterations of thyroid epithelial cells observed in these thyroids were not believed to be related directly to irradiation. These studies indicated that amelioration of hyperthyroidism may occur after irradiation of the thyroid gland with I^{131}, usually in the absence of the classic structural changes generally associated with injury from irradiation.

Journal of Infectious Diseases, Chicago

93:207-302 (Nov.-Dec.) 1953. Partial Index

Isolation of West Nile Virus in Israel. H. Bernkopf, S. Levine and R. Nerson.—p. 207.
Isotopic Studies of Fluid and Electrolyte Changes in Domestic Rabbits with Rocky Mountain Spotted Fever. J. K. Aikawa and G. T. Harrell. —p. 222.
Antigenic Studies of Group of Paracolon Bacteria. W. C. Eveland and J. E. Faber Jr.—p. 226.
Effect of Meningopneumonitis Virus Infection on Glycolysis by Mouse and Rat Brain. A. H. Fieldsteel and W. S. Preston.—p. 236.
Concept of "Direct Antagonism" in Bacteria. E. S. Wynne and J. O. Norman.—p. 243.
Changes in Tissue Radiosodium Space Associated with Experimental Rocky Mountain Spotted Fever in Guinea Pigs. J. K. Aikawa and G. T. Harrell.—p. 263.
Antigenic Hemophilus Pertussis Vaccines Grown on Charcoal Agar. P. W. Ensminger, C. G. Culbertson and H. M. Powell.—p. 266.
Further Observations on Effect of 2,4-Dinitrophenol on Growth of Influenza Virus. M. D. Eaton and M. E. Perry.—p. 269.
Studies in Old Mice to Test Hypotheses of Local and General Immunity to Trichinella Spiralis. J. E. Larsh Jr.—p. 282.
Russian Spring-Summer Encephalitis and Louping Ill Group of Viruses: Relationship of European and Asiatic Strains of Russian Spring-Summer Encephalitis Viruses and Louping Ill Virus. W. L. Pond, S. B. Russ and J. Warren.—p. 294.

Journal of International College of Surgeons, Chicago

20:667-800 (Dec.) 1953

Newer Trends in Treatment of Old Congenital Dislocations of Hip. J. Hass.—p. 667.
Replacement of Pericardium: Experimental Work with Vinyon H H Staple in Dogs. J. Costas-Durieux, R. Weller and C. P. Bailey.—p. 682.
*Acute Mesenteric Arterial Occlusion: Restoration of Blood Flow by Embolectomy. A. A. Klass.—p. 687.
*Enzymatic Débridement of Suppurations, Necrotic Lesions and Burns with Papain. A.-V. Guzman and M. G. Stein de Guzman.—p. 695.
20 Years' Experience with Myomectomy During Childbearing Period. G. Jones.—p. 703.
Surgical Treatment of Bilateral Lithiasis. S. Petkovic.—p. 709.
Treatment of Slipping of Upper Femoral Epiphysis. B. Howorth.—p. 716.
Problems Encountered in Fenestration. G. E. Shambaugh Jr.—p. 736.
Recurrent Dislocation of Shoulder. P. Leguit.—p. 741.
Monocytic Leukemia: Oral and Anorectal Involvement: Report of Case. M. M. Marks.—p. 750.
Retromesocolic Hernia: Development and Treatment. P. J. Burnham. —p. 753.
Conservative Therapy of Carotid Body Tumors. E. L. Doermann and W. H. Meffley.—p. 761.
Three Minutes in Cardiac Arrest. W. A. Barrett, J. R. Green, A. N. Heinrich and H. Uram.—p. 765.
Hepato-Enterostomy. I. Honjo and C. Araki.—p. 775.
Congenital Right Diaphragmatic Hernia in an Adult. H. F. Mars and J. A. Bollinger.—p. 780.

Occlusion of Mesenteric Artery.—Acute occlusion of the superior mesenteric artery is reported in a 64-year-old man and in a 67-year-old woman. The occlusion was correctly diagnosed in both patients, and surgical removal of an obstructing intra-arterial plug was carried out successfully, although both

patients died within 60 and 56 hours, respectively, after the operation. Death of the man resulted from myocardial failure, and the woman died of massive intraperitoneal hemorrhage from mesenteric veins that had been cut in the dissection necessary to expose the superior mesenteric artery in the course of the operation. Necropsy in both cases revealed that the bowel was normal and in a condition consistent with survival. In the man the bowel had been totally deprived of blood for 14 hours and in the woman for 12 hours. In both patients, the superior mesenteric artery and its branches remained patent after removal of the obstructing lesion. The author makes several conclusions. 1. Circulation in the superior mesenteric artery and its branches can be restored by removal of the obstructing thrombus. 2. Circulation within the bowel wall compatible with continuing viability can be assured by this procedure. 3. The bowel wall can survive total deprivation of blood for a period of 14 hours. Nothing can be said with reference to survival beyond this time limit, but parallel experience with muscle tissue generally would indicate that it cannot safely be exceeded. 4. During the operation, particular care should be taken to secure arrest of hemorrhage, which may occur after restoration of the mesenteric blood flow. This applies particularly to the dissection of mesenteric fat that is necessary to expose the superior mesenteric artery. 5. The postoperative use of heparin should be avoided. Sufficient reliance can be placed on flow velocity in an artery to prevent reformation of a thrombus at the site of the incision into the vessel.

Débridement with Papain.—The fibrinolytic enzyme papain, the dried milky juice of the melon tree Carica papaya, was given a therapeutic trial in the débridement of chronic suppurative and necrotic areas of 13 children and 7 adults with second and third degree burns, chronic leg ulcers, gangrene, carbuncle, chronic suppuration, and dry crusts. Several commercial preparations of papain were used that varied greatly in potency. In three patients, the fibrinolytic potency of the enzyme was compared with that of a standard preparation of streptokinase-streptodornase and was observed to be similar. Two methods were employed for the treatment: (1) direct application of the powder with a common vaginal insufflator, and (2) application of wet dressings with freshly prepared solutions of the enzyme, ranging from 2 to 5%. Both methods proved effective provided that the lesion and the dressings were prevented from drying within a minimum period of 24 hours by using several layers of surgical gauze well soaked with isotonic sodium chloride solution and a final layer of petrolatum gauze. Papain did not cause any deleterious action on healthy tissues, and systemic or generalized reactions were not noticed during its application. The powder and solutions used were not bacteriologically sterile and, nevertheless, no reaction attributable to their application was observed. The cost of papain is so low that any hospital or patient can afford to use it extensively.

Journal Lab. and Clinical Medicine, St. Louis

42:833-1006 (Dec.) 1953

Uptake in Vitro of Radioactive Iron by Avian Erythrocytes. W. N. Jensen, H. Ashenbrucker, G. E. Cartwright and M. M. Wintrobe. —p. 833.
Interference with Fibrin Formation in Multiple Myeloma by an Unusual Protein Found in Blood and Urine. C. G. Craddock Jr., W. S. Adams and W. G. Figueroa.—p. 847.
Intrinsic Factor Studies: II. Effect of Gastric Juice on Urinary Excretion of Radioactivity After Oral Administration of Radioactive Vitamin B_{12}. R. F. Schilling.—p. 860.
Distribution of Colloidal Radioactive Chromic Phosphate After Intracavitary Administration in the Rat. J. A. D. Cooper and E. M. Zorn. —p. 867.
Studies of Liver Function Tests: IV. Effect of Repeated Injections of Sodium Benzoate on Formation of Hippuric Acid in Patients with Liver Disease. L. Zieve and M. Hanson.—p. 872.
Antibiotic Combinations and Resistance to Antibiotics: Development of Resistance During Repeated Subcultures of Staphylococci and Certain Streptococci on Media Containing Penicillin, Streptomycin, Erythromycin, Terramycin, and Chloramphenicol Used Singly and in Pairs. S. S. Wright, E. M. Purcell, C. Wilcox and others.—p. 877.
Serologic Diagnosis with Respect to Histoplasmosis, Coccidioidomycosis, and Blastomycosis and Problem of Cross Reactions. C. C. Campbell and G. E. Binkley.—p. 896.

Maine Medical Association Journal, Portland

44:299-326 (Nov.) 1953

Psychosomatic Aspects of Medicine. D. W. Atchley.—p. 299.
Temporal Arteritis: Treatment with Cortisone: Two Case Reports. M. A. Chapin and R. A. Frost.—p. 304.
Cardiac Lesions in Rheumatoid Arthritis. C. W. Steele.—p. 308.
Hyperleukocytosis and Macrocytic Anemia Resulting from Carcinoma of Ileum: Case Report. M. A. Chapin.—p. 312.
Modified Pomeroy Sterilization. E. T. Bennet.—p. 314.

44:327-358 (Dec.) 1953

*Subendocardial Fibroelastosis. L. E. Morris Jr. and F. C. Emery.—p. 327.
The Family Physician and Cardiac Surgery. W. B. Manter.—p. 331.
Regional Ileitis. R. T. Munce.—p. 334.
Instrumental Management of Forecoming Head. W. M. Shubert.—p. 336.
Hyaline Membrane Disease. S. E. Monaghan.—p. 338.

Subendocardial Fibroelastosis.—The infant whose case is reported had been born at term, but the mother had had a moderate toxemia of pregnancy. Physical examination at birth and subsequent neonatal course suggested that the child was normal. The infant was seen again at 3 months of age because of anorexia and irritability. Examination showed anemia. There was a good clinical response to administration of iron, liver, and vitamin B_{12}. One week prior to admission the patient seemed to have a "cold" with nasal discharge, cough, and return of anorexia and irritability. Rapid, irregular respirations were noted, and the infant refused food and seemed to cry continually. The respirations were rapid and grunting in type. The cyanosis became worse on crying. There was lack of breath sounds with an impaired resonance over the left chest. The heart sounds were indistinct. The diagnosis was pneumonia with atelectasis and pleural effusion of the left lung. The patient was placed in oxygen and given penicillin and streptomycin parenterally. By the second hospital day, there was no improvement in respiration, but cyanosis was no longer present and pneumonia seemed unlikely. On the third day, antibiotics were discontinued. The infant became more distressed without oxygen and gave indications of being in severe pain. A diagnosis of subendocardial fibroelastosis was considered as well as a possible anomalous origin of the left coronary artery. On the fifth hospital day the condition was worse, and congenital heart disease was thought of. On the ninth day signs of decompensation developed and digitalis was started. A pericardial tap was done to relieve possible tamponade but no fluid was found. The patient exhibited a brief period of Cheyne-Stokes respirations and died on the 13th hospital day. Autopsy revealed dilation of the right auricle. The heart weighed 88 gm. On microscopic examination, sections of the interventricular septum showed endocardial thickening due to proliferation of loose, basophilic tissue. Toward the myocardium, this tissue became progressively more dense and collagenous. The lungs showed atelectasis. Two types of endocardial thickening may be recognized; primary and secondary. The latter is a reparative fibrosis and may be found in such diseases as myocardial infarction, rheumatic fever, diphtheria, subacute bacterial endocarditis, malignant nephrosclerosis, periarteritis nodosa, disseminated lupus erythematosus, and beriberi. The primary type may be found at any age but it is commoner in infants and children. The symptoms in the reported case were similar to those of previous reports, but cardiac pain seemed severer. In children this disease is best explained as a developmental anomaly, perhaps an overgrowth of the endocardium of the left bulbus cordis. It may also be secondary to a valvular or other cardiac defect leading to anoxia of the endocardium. In adults, the etiology and pathogenesis remain obscure. Rapid myocardial failure in a previously healthy person, with or without peripheral embolization, should suggest this disorder.

Medical Annals of District of Columbia, Washington

22:639-708 (Dec.) 1953

Physical Measurement and Medicine. A. V. Astin.—p. 639.
Vaccination Against Influenza. D. J. Davis.—p. 643.
Agammaglobulinemia (Congenital Absence of Gamma Globulin): Report of Case. O. C. Bruton.—p. 648.
Rheumatoid Arthritis-Like Syndrome During Apresoline Therapy: Report of Case. V. P. Beelar.—p. 651.
Depression: Some Dynamic and Clinical Features of Depressive Character Defenses, Psychoneurotic Depression and Suicide. H. P. Laughlin. —p. 653.

Michigan State Medical Society Journal, Lansing

52:1137-1256 (Nov.) 1953

Newer Concepts Concerning Tuberculosis During Childhood. M. I. Levine.—p. 1169.
Anesthesia Accidents. J. S. Lundy.—p. 1174.
Psychiatry for the General Practitioner. H. A. Luce.—p. 1177.
Methostan in Treatment of Nutritional Dwarfism: Preliminary Report. W. H. Hewes.—p. 1181.
Hyperthyroidism: Rationale of Modern Treatment. C. W. Eberbach. —p. 1189.
Intestinal Obstruction—1900 vs. 1952. M. O. Cantor and R. P. Reynolds. —p. 1194.
Diagnostic Problems of Cancer. R. W. Houde.—p. 1197.
Observations on Diagnosis and Treatment of Varicose Veins. J. G. Slevin.—p. 1201.
Our Heritage in British Medicine from Bright to Gull. R. M. McKean. —p. 1204.
Severe Urticaria and Angio-Edema Resulting from Phenylbutazone (Butazolidin®). S. J. Levin.—p. 1210.

New England Journal of Medicine, Boston

249:871-914 (Nov. 26) 1953

The Balanced Curriculum. H. Cohen.—p. 871.
Value of Single Injection of Thyrotropin in Diagnosis of Obscure Hypothyroidism. W. M. Jefferies, R. P. Levy, W. G. Palmer and others. —p. 876.
Fractures of Carpal Navicular: Accurate Diagnosis and Planned Treatment. E. C. Branson.—p. 884.
Respiratory Depressant Action of N-Allylnormorphine in Normal Subject and in Patients with Respiratory Acidosis Secondary to Pulmonary Emphysema. S. M. Tenney and J. C. Mithoefer.—p. 886.
War Wounds. E. J. Pulaski.—p. 890.
*Treatment of Pediculosis Ciliorum in Infant. F. Ronchese.—p. 897.

249:915-956 (Dec. 3) 1953

A Rededication? J. P. Bowler.—p. 915.
Unrecognized Emboli to Lungs with Subsequent Cor Pulmonale. W. R. Owen, W. A. Thomas, B. Castleman and B. F. Bland.—p. 919.
Pilonidal Sinus: Experience with 449 Cases. R. W. Dwight and J. K. Maloy.—p. 926.
Cholecystography in Presence of Liver Disease. A. D. Etess and B. Straus.—p. 930.
War Wounds. E. J. Pulaski.—p. 932.
Neurofibroma of Diaphragm. R. H. Sweet and T. Gephart.—p. 939.

Treatment of Pediculosis Ciliorum in Infants.—According to Ronchese pediculosis (infestation with Phthirus pubis or crab lice) of the eyelashes and the edges of the eyelids is rare in adults but less rare in infants, who become infested by passage of the lice from the axillary or chest hair of the parents or attendants. The lice are visible to the naked eye or can be seen with the help of magnifying glasses. Treatment is simple; the lice and ova (nits) are easily picked out with a pair of cilia forceps (tweezers) or trachoma forceps (not just any forceps). The removal of the nits is facilitated by the infant's tears. In cases of such long standing that acute or chronic blepharitis has developed, antiseptic or antibiotic medication may be indicated. In early uncomplicated cases removal of the lice and nits is sufficient and medicaments are unnecessary. Recurrences are due to reinfestations from the same source, parents or attendants.

249:957-1004 (Dec. 10) 1953

*Mallory-Weiss Syndrome: Hemorrhage from Gastroesophageal Lacerations at Cardiac Orifice of Stomach. J. P. Decker, N. Zamcheck and G. K. Mallory.—p. 957.
Hindquarter Amputation: Experience with 18 Cases. G. W. Taylor and W. P. Rogers Jr.—p. 963.
Use of Radioactive Iodine in Detection of Thyroid Dysfunction. W. W. Drummy Jr.—p. 970.
Acute Hemolytic Anemia Associated with Infectious Mononucleosis. B. D. Hall and F. C. Archer.—p. 973.
Dermatologic Therapy. J. G. Downing.—p. 976.
*Peripheral Vascular Collapse After Subcutaneous Use of Hypertonic Nonelectrolyte Solution. J. J. Butler.—p. 988.

Hemorrhage from Gastroesophageal Lacerations.—Decker and his associates say that since the original descriptions of gastrointestinal hemorrhage caused by cardioesophageal lacerations induced by vomiting presented by Mallory and Weiss more than twenty years ago, there have been no further reports of this syndrome. Several large series of gastrointestinal bleeding recently studied from a clinical standpoint make no mention of this syndrome as a cause for hematemesis. However, among 101 patients with gastrointestinal bleeding, recently examined at autopsy at the laboratory with which these authors are connected, 3 were found in whom death was caused by cardioesophageal lacerations. At least 11 cases of gastrointestinal hemorrhage due to cardioesophageal lacerations have been examined at autopsy in their laboratory. Clinical and pathological observations in these cases and six case histories illustrative of various features of the syndrome are reported. Examination of the case histories, in an attempt to clarify the pathogenesis of these lesions, revealed that the common factor was vomiting. Analysis of the cases yielded no explanations more reasonable than that advanced by Mallory and Weiss—namely, that the lacerations were produced by violent retching movements, which forced the gastric contents against a contracted cardiac sphincter. Reports of cases of spontaneous rupture of the esophagus emphasize the importance of violent retching and vomiting. The similarity of these conditions suggests a common mechanism. Atrophic gastritis is a possible underlying factor in the production of lacerations at the cardia. Atrophic mucosa may be less resistant than normal mucosa to the traumatic effects of vomiting. A significant degree of atrophic gastritis was present in six of the eight cases in which specimens of gastric mucosa were available for microscopic study. In animals, gastritis with ulceration and bleeding can be produced by a variety of nutritional deficiencies, particularly of components of the vitamin B complex. Alcoholism is commonly accompanied by vitamin deficiencies, particularly of the C and the B group. Most of the cases of this syndrome originally reported by Mallory and Weiss were associated with alcoholism. Many of the cases in the present series, however, were associated with a variety of diseases not only in the gastrointestinal tract but also in remote organs. Protracted vomiting or retching remains the common etiologic factor in all cases. Associated pathogenetic factors include chronic atrophic gastritis, alcoholism and nutritional cirrhosis.

Peripheral Vascular Collapse After Subcutaneous Use of Hypertonic Nonelectrolyte Solution.—Butler presents a case in which 1,200 cc. of hypertonic nonelectrolyte solution was given subcutaneously on the second day of an uneventful postoperative period. Severe peripheral collapse resulted. The patient responded well to administration of plasma and whole blood. The author cites experimental observations and a warning editorial on the peripheral vascular collapse that may be induced when hypertonic nonelectrolyte solutions are injected subcutaneously. The collapse can probably be explained by the shift of fluid and electrolytes from one body compartment to another. Subcutaneous infusions should probably be restricted to the use of isotonic electrolyte solutions.

New Jersey Medical Society Journal, Trenton

50:487-534 (Nov.) 1953

*Selection of Cases for Mitral Commissurotomy. N. A. Antonius, R. Miller, H. Green and A. D. Crecca.—p. 490.
Medical Degrees in New Jersey. F. B. Rogers.—p. 496.
Infarction of Liver Due to Polyarteritis Nodosa. H. P. Price and C. J. Flanagan.—p. 500.
Asthma in First Year of Life. W. P. Buffum.—p. 503.
*Iatrogenic Meningitis. M. Cutler and P. Cutler.—p. 510.
Idiopathic Hemorrhagic Infarction of Testis in Newborn. A. R. Fernicola. —p. 515.
Organic Diseases Simulating Psychogenic Disorders. A. D. Dennison Jr. —p. 520.

Mitral Commissurotomy.—As a result of their experiences with 100 patients operated on for mitral stenosis, Antonius and co-workers subscribe to a compromise view between the extreme recommendations of two surgical schools that consider for mitral commissurotomy those patients with dynamically significant mitral stenosis in whom early symptoms have appeared and progressive congestive heart failure has not developed. Contraindications to surgical intervention consist of active rheumatic infection, subacute bacterial endocarditis, significant aortic valvular lesions, pronounced mitral insufficiency, pronounced cardiomegaly with extensive myocardial damage, emotional instability, intractable heart failure, or associated hypertensive or coronary heart disease. History and physical examination, fluoroscopy, electrocardiography, and cardiac catheterization may be utilized in arriving at the conclusion regarding suitability for surgical intervention. The underlying physiology of mitral stenosis determining the rationale for

such surgical intervention is outlined. Surgical repair of the deformed valve is but an adjunct in the care of rheumatic cardiac patients; they may be restored to great activity, enjoying a more normal life, yet they must, for the immediate future, be considered as cardiac patients under the continued supervision of their physicians.

Iatrogenic Meningitis.—Two cases of meningitis resulting from lumbar puncture for spinal anesthesia, one in a 19-year-old woman and one in a 60-year-old man, are reported with an extensive review of the literature. Iatrogenic meningitis is increasing. Lumbar puncture for diagnosis, anesthesia, or therapy is responsible for most cases. Routes of bacterial contamination are the anesthetic solution, unsterile instruments, supposedly sterile water used in rinsing syringes and needles, "sterile jars" used in storing syringes, air, and vials of penicillin used for intrathecal medication. Culture of the spinal fluid of both of the authors' patients revealed Pseudomonas aeruginosa, the causative agent in most cases of iatrogenic meningitis. Examination of the spinal fluid with ultraviolet light is a simple, rapid clinical means of identifying this organism. The diagnosis of iatrogenic meningitis is usually simple to make. A history of lumbar puncture followed in 12 hours to 10 days by meningitic signs establishes the diagnosis. This type of meningitis, in contrast to the epidemic meningococcic type, tends to be subacute only in its intensity; the natural course of the disease is long and is characterized by remissions and relapses. Exacerbations may occur even months after an apparent cure. Penicillin not only was ineffective, but may actually stimulate growth of Pseudomonas. It should not be administered to patients with iatrogenic meningitis. Of 23 cases collected from the literature in which streptomycin was given either intrathecally, parenterally, or combined, 15 were cured and 8 died. In the authors' female patient, Pseudomonas aeruginosa was resistant to penicillin, bacitracin, streptomycin, and chlortetracycline (Aureomycin), but was moderately sensitive to both oxytetracycline (Terramycin) and chloramphenicol (Chloromycetin); a complete cure was obtained with the two latter drugs. Recovery of the man was obtained with streptomycin given by intramuscular and intrathecal routes. Polymyxin was effective in the few reported trials. Neomycin proved effective in two cases. Effective therapy depends on proper identification and sensitivity tests of the organism involved.

New York State Journal of Medicine, New York

53:2415-2550 (Nov. 1) 1953

Value of Estrogen Therapy in Obstetric Difficulties and Puerperium. T. Neustaedter.—p. 2455.
Significance of Abnormal Uterine Bleeding Due to Ovarian Dysfunction. I. Frost.—p. 2459.
Use of ACTH and Cortisone Therapy in Obstetrics and Gynecology. G. S. Jones and J. E. Howard.—p. 2463.
*Advantages and Risks of Preserving Ovary After Age of 45. C. L. Randall and P. R. Gerhardt.—p. 2467.
Indications and Complications of Recession Operation on Inferior Rectus Muscle. H. W. Brown.—p. 2472.
Modern Trends in Cataract Surgery. T. LeWin.—p. 2479.
Ocular Changes in Head Injuries. J. F. Gipner.—p. 2481.
Treatment of Chronic Discharging Ear. J. A. Sullivan.—p. 2486.
Malignancies of Ear, Nose, and Throat. D. W. Brewer.—p. 2492.
Statistical Study of Postoperative Tonsil and Adenoid Hemorrhage. R. J. Bellucci.—p. 2495.
Psychosomatic Aspects of Educated Childbirth. H. B. Davidson.—p. 2499.
Some Psychologic Observations of "Educated Childbirth." P. Donovan and S. Landisberg.—p. 2504.
Thyroid Cancer. W. Trevor.—p. 2511.
Pattern of Skin Eruption Due to Fleas. L. Wirth.—p. 2513.

Advantages and Risks of Preserving Ovary After Age 45.—The advantages of preserving the ovaries when operating on women over 45 are often regarded as not sufficiently important to warrant the risk that a malignant lesion may develop if an ovary is not removed at the time of hysterectomy. Physicians and laboratories in the state of New York for the past 12 years have been reporting all malignant tumors to the Division of Cancer Control. From the information thus obtained the authors estimate that the calculated risk of preserving the ovary, at least in women "over fifty," would appear to be that, in 1,000 women subjected to pelvic laparotomy, in 8 a malignant and in 6 a benign neoplasm will develop, all of which could have been prevented if both ovaries had been removed at laparotomy. They doubt that this warrants the removal of normal-appearing ovaries merely as a prophylactic measure. The chance of bilateral involvement, not grossly appreciable but already developing in the other ovary, does indicate careful inspection and bisection of the opposite ovary before it can be regarded as normal and uninvolved by a neoplasm involving one side. When contemplating the prophylactic removal of ovaries in women over 45, account should be taken of two facts: In a review of over 1,100 benign cystomas it was found that approximately 70% had developed before the age of 45, and the probability of a malignant lesion developing in a woman's ovary is now known to decrease rapidly after her 50th year.

North Carolina Medical Journal, Winston-Salem

14:593-648 (Dec.) 1953

Prevention of Rheumatic Fever. E. Craige.—p. 593.
Nutrition in Obstetrics. R. L. Burt.—p. 596.
Bronchial Arteries: Review of Their Physiology in Health and Disease. C. B. Taylor.—p. 603.
Recent Advances in Treatment of Infantile Hydrocephalus. E. Alexander Jr. and C. H. Davis Jr.—p. 610.
Breast Carcinoma: Appraisal of Surgical Treatment. C. H. Ingram. —p. 614.
Radical Radiotherapy in Breast Cancer: Analysis of Modified McWhirter's Technique. F. R. Gilmore.—p. 617.
Diagnosis and Management of Poisoning Due to Organic Phosphate Insecticides. H. R. Chamberlin.—p. 623.
Treatment of Pyogenic Granulomas and Hemangiomas. F. M. Hodges. —p. 628.

Ohio State Medical Journal, Columbus

49:1061-1172 (Dec.) 1953

Treatment of Chest Injuries. D. K. Heydinger and K. P. Klassen. —p. 1081.
*Unlimited Diet in Peptic Ulcer—1,000 Cases. E. A. Marshall.—p. 1085.
Sex Hormone Therapy in Gynecology. E. Eichner.—p. 1089.
Day Hospital Unit—Milieu Therapy, Its Place in Treatment of the Mentally Ill. P. C. Rond.—p. 1093.
Use of Hydrocortisone-Neomycin Ointment. R. L. Kile.—p. 1097.
Treatment of Pyogenic Skin Infections with Ilotycin® Ointment (Erythromycin). I. L. Schonberg.—p. 1099.

Unlimited Diet in Peptic Ulcer.—It was Marshall's aim to set up a schedule that would permit the ulcer patient to eat and drink everything without distress at the end of 24 hours. While this can be done in many ways, the author feels that the use of combination tablets is the simplest method. Aluminum hydroxide, because of its adsorptive properties, was the basic treatment in most of the 1,000 patients treated by the author. A no. 2 tablet, which contained calcium carbonate, 0.5 gm., magnesium oxide, 0.25 gm., phenobarbital, 0.016 gm., and atropine, 0.0002 gm., was compounded to furnish neutralization and sedation and to combat the constipating effect of aluminum hydroxide. This tablet was effective, but had a somewhat excessive laxative effect, and so in a no. 3 tablet the magnesium oxide was dropped and the calcium carbonate increased to 0.65 gm. With the use of the three tablets—first, a 0.65 mg. tablet of aluminum hydroxide and tablets or capsules no. 2 and no. 3—the schedule is usually set up on an ambulatory basis. Assuming that the patient arises at 6:30 a. m. and retires at 11 p. m., he is given two tablets (1.3 gm.) of aluminum hydroxide at 6:30, 7:30, 8:30, etc., every hour on the half-hour until 10:30 p. m. Then, at 11 p. m., 1 a. m., and 3 a. m. the aluminum hydroxide dose is increased to four tablets (2.6 gm.) and, unless milk allergy is present, 240 cc. of whole milk is given in addition on each of these three hours. As to the no. 2 and no. 3 tablets, the daily dose is nine tablets crushed (or capsules). Usually with the above 44 tablets (28.6 gm.) of aluminum hydroxide, the schedule will call for six no. 2 and three no. 3 tablets, that is, one no. 2 at 7 and 9 a. m. and 1, 3, 7 and 9 p. m. with one no. 3 at 11 a. m. and 5 and 11 p. m. with the first night feeding if bowel habits continue to be normal. The author stresses that the more frequent the medication the better and warns that the dose and frequency should not be cut for a mininum of four and one-half months. Unlimited diet was successfully given in 921 of the 1,000 ulcer patients treated. The advantages of this treatment include the

following factors. 1. It presents immediate evidence of inadequate therapy. 2. It permits a more nutritious diet. 3. Because of the rapid disappearance of the niche under heavier therapy, the differentiation of malignant from benign gastric ulcer is aided. 4. Separation of surgical from medical hemorrhagic cases is accomplished in 24 hours. A patient who bleeds on "heavy" therapy is not likely to stop on further medical treatment. 5. Over 95% of the obstructed, nonstenotic cases may be salvaged and the surgical cases are apparent immediately. 6. Ulcer patients like to eat and unlimited diet adds to their security. 7. Serious complications are diminished when this method is followed. 8. The period between cessation of therapy and recurrence is greatly extended. The fact that the average ulcer patient was seen by his physician about every six weeks, or about three or four times, and the long symptom-free period greatly diminishes the cost of treatment of peptic ulcer.

Postgraduate Medicine, Minneapolis

14:465-562 (Dec.) 1953. **Partial Index**

Place of Antihistaminics in Treatment of Allergic Disorders. J. M. Sheldon.—p. 465.
Metastatic Manifestations as Presenting Symptoms of Primary Cancer of Lung. S. A. Leader and R. J. Borgerson.—p. 470.
Evaluation of Present-Day Treatment of Peptic Ulcer. H. M. Pollard. —p. 481.
Postphlebitic and Varicose Vein Stasis. W. J. M. Scott.—p. 494.
Transverse and Oblique Muscle-Splitting Abdominal Incisions. L. T. Palumbo and I. A. Katz.—p. 503.
Use of Nylon Mesh in Repair of Abdominal Wall Defects. B. B. Landry. —p. 510.
Value of Operative Cholangiography. A. Behrend and B. Greenspan. —p. 514.
Transthoracic Dorsolumbar Sympathectomy. W. P. Kleitsch.—p. 519.
Acute Nonbacterial Gastroenteritis. R. G. Hodges.—p. 525.
*Treatment of Acute Bacterial Meningitis in Infants and Children. M. H. D. Smith and G. W. Herring.—p. 540.
Pediatric Responsibility to Children with Speech Defects. H. Bloomer. —p. 546.

Treatment of Acute Bacterial Meningitis.—The study reported by Smith and Herring was designed to evaluate the results obtained when all patients with "purulent meningitis" were treated according to the same regimen, regardless of causative organism. Bacteriological studies were carried out so that it would be possible in retrospect to evaluate the treatment in each type of meningitis. Of the 98 patients with meningitis admitted to the contagious disease service of the Tulane Pediatric Department at Charity Hospital, 86 were included in the present analysis. On admission a lumbar puncture was performed and the pressure was determined; spinal fluid was obtained for smear, culture, cell count, and chemical analysis. The patient was then given sodium sulfadiazine intravenously (50 mg. per kilogram of body weight) and aqueous penicillin (200,000 units intravenously). At the same time sulfadiazine was given by mouth or by stomach tube (100 mg. per kilogram of body weight), also chloramphenicol (100 mg. per kilogram of body weight). Subsequent medication consisted of sulfadiazine (150 mg. per kilogram of body weight daily, divided into four or six doses); chloramphenicol (250 mg. every 6 or 8 hours, depending on the size of the child); and aqueous penicillin (200,000 units intramuscularly every 8 hours). Treatment was continued for a minimum of six days or until the patient had been afebrile for 48 hours, whichever was longer. All of the patients were less than 10 years old, and 51, or considerably over half, of the 86 children were less than 2 years old; that is, they belonged to the age group in which mortality is likely to be highest. Seven of the 86 children died (8.1%). Bacteriological studies showed that in 32 patients Hemophilus influenzae was the causative agent, in 26 the meningococcus; in 14 the pneumococcus. In 14 the causative agent could not be identified. Analysis of the data suggests that, where reliable bacteriological help is not available, the above three drugs in combination may be used in the treatment of any child, more than 2 to 3 months of age, with purulent meningitis with expectation of a satisfactory outcome. An unusually high incidence of complications in patients with H. influenzae meningitis who were treated with the palmitate ester of chloramphenicol suggests that this compound is to be avoided in children who have severe, acute infections.

Proc. Staff Meet., Mayo Clinic, Rochester, Minn.

28:673-704 (Dec. 2) 1953

SYMPOSIUM ON MIGRAINE

*Migraine: Diagnosis and Treatment with Emphasis on Migraine-Tension Headache, Provocative Tests and Use of Rectal Suppositories. G. A. Peters.—p. 673.
Ocular Manifestations of Migraine: Report of 4 Cases of Hemianopsia. R. W. Hollenhorst.—p. 686.
Some Psychiatric Aspects of Migraine. M. E. Giffin.—p. 694.
Male Hypogonadism: III. The Testis in Pituitary Dwarfism. A. Albert, L. O. Underdahl, L. F. Greene and N. Lorenz.—p. 698.

Diagnosis and Treatment of Migraine and Tension Headache.—Periodicity and recurrence, positive family history, cortical phenomena, gastrointestinal disturbance, unilateral nature as well as shifting location of pain in the head, and positive response to ergotamine products indicate a diagnosis of migraine. If most or all of these characteristics are present, the migraine is typical, but oftener it appears in an atypical form. Typical migraine has three vascular phases, namely (1) vasoconstriction, (2) vasodilatation, and (3) edema. During the usually short (half hour), prodromal phase vasoconstriction involving the intracranial arteries occurs and produces scotoma, hemianopsia, paralysis, diplopia, paresthesia, and aphasia. During vasodilatation, headache is experienced, and associated symptoms are anorexia, nausea, vomiting, diarrhea, dizziness, fever, palpitation, and excessive perspiration and urination. There may be flushing or pallor of the face, reddening of the conjunctivas, and nasal congestion. Although the stage of edema is not commonly seen, when observed, it is characterized by swollen, hard, and tender extracranial vessels on the side of the headache. The throbbing headache of the vasodilating phase evolves into a constant sharp pain with tenderness, usually in the frontal, temporal, and parietal regions, that lingers until the attack has subsided. During this phase vasoconstrictive agents are not only ineffective but may aggravate the pain. The history is the most important diagnostic aid, but provocative tests with nitroglycerin sublingually, histamine subcutaneously, or nicotinic acid are helpful. "Tension headache" is a pain in the head arising from "tension," a state of nervous hyperirritability, which results from emotional stimulation or conflict. Migrainous patients often say, "I have two kinds of headache" and describe the dull, generalized headache of tension type and a severer type that comes periodically, perhaps once or twice a week or month. This is throbbing, unilateral (although it can be bilateral), and associated with nausea, photophobia, and perhaps vomiting. The migraine headache responds to ergotamine products, while the tension headache will not. Migraine is almost always associated with nervous tension. Drug therapy is usually enough to abort a mild attack, but, when moderate or severe, the migrainous attack becomes resistant to drug therapy and at times nothing is effective except sleep-inducing agents. The different vascular phases of migraine must be considered during treatment. Thus, during the early vasoconstriction, vasodilators such as nicotinic acid, in doses of 100 to 200 mg. by mouth, may abort the cortical phenomena, or inhalation of 10% carbon dioxide with 90% oxygen may be used, if nicotinic acid is not effective. During vasodilatation the vasoconstricting drugs are most important. However, many patients will get relief from simple measures such as aspirin or rest. One of the best vasoconstricting preparations is cafergot, a tablet containing 100 mg. of caffeine alkaloid and 1 mg. of ergotamine tartrate. For severer attacks, dihydroergotamine is recommended. Patients who have migraine as well as tension headaches must learn to rest and relax, to avoid fatigue. While migraine cannot be cured, much relief can be provided.

Southwestern Medicine, El Paso, Texas

34:433-480 (Dec.) 1953

Use of Anticoagulant Phenylindanedione in Ambulatory Patients. F. W. Drinan, H. Sise and W. C. Moloney.—p. 450.
Rheumatic Activity in Patients with Mitral Stenosis Undergoing Valvuloplasty: Pathological Aspects. J. P. Decker, C. V. Hawn and S. L. Robbins.—p. 461.
Id.: Clinical Aspects. W. F. McNeely.—p. 461.
Diagnosis of Mitral Regurgitation. W. H. Abelmann.—p. 462.
Peyronie's Disease (Plastic Induration of Penis or Fibrous Cavernositis). R. F. Thompson.—p. 463.

FOREIGN

Archiv für Kinderheilkunde, Stuttgart

147:209-312 (No. 3) 1953. **Partial Index**

Contribution to Therapy of Hemorrhagic Internal Pachymeningitis (Hematoma Durae Matris). F. Koch.—p. 213.
Pathogenesis of Adrenogenital Syndrome. J. R. Bierich and C.-D. Voigt. —p. 225.
Present Status of Infectious Diseases. H. Zischinsky.—p. 237.
Problems of Parotitis; Mumps Meningitis and Significance of Diastase in Mumps. H. Zischinsky and E. Hausmann.—p. 244.
*Chylous Ascites in Infants in Course of Primary Intestinal Tuberculosis. H. Beyer.—p. 249.

Chylous Ascites in Infants with Primary Intestinal Tuberculosis.—The occurrence of chylous ascites is reported in a 5½-month-old baby boy in whom primary intestinal tuberculosis was diagnosed on roentgenologic examination. Severe inflammatory (tuberculous) changes of the small intestine were observed. Multiple intra-abdominal calcifications demonstrated the extensive involvement of the lymph nodes of the mesentery. Chyle stasis may be considered in addition to inflammatory changes of the walls of the chyliferous vessels (tuberculous lymphangitis). An inflammatory process was suggested in the author's case and in other cases collected from the literature by the fact that recovery resulted from repeated abdominal puncture or pneumoperitoneum. Both procedures are not to be considered as causal treatment. The abdominal puncture in the author's case probably stimulated the peritoneum in the sense of absorption besides causing mechanical discharge. The protein values of the ascitic fluid, which initially were higher than those in the blood, with an increased amount of globulin, also favored the concept of an inflammatory component. In the retroperitoneal space, which may be assigned a key position in the causation of the chylous ascites, are seen converging (1) the lymph vessels of the lower extremities and the pelvic organs; (2) the lymph vessels of a major portion of the deeper layers of the abdominal wall; (3) the lymph vessels of the kidneys, adrenals, and the inferior surface of the diaphragm; (4) the lymph vessels of the stomach, spleen, pancreas, and liver, which are connected with the celiac lymph nodes; and (5) the chyle vessels of the intestine. The lymph nodes of the retroperitoneal space form a chain with the lymph vessels that run toward the cranium. This chain passes along the vena cava inferior and the abdominal aorta and is connected with the iliac-lumbar lymph nodes that pass along the iliac artery and vein. These anatomic relations explain some of the idiopathic cases reported in the literature. The author believes that the extent of the participation of the chyliferous vessels in the distal portions of the ileum-jejunum segment is so extensive that a "collateral circulation" does not become effective because of the involvement of numerous anastomoses of the lymph system in the mesentery, or that the exudation from the chyliferous vessels must be sufficient for the production of a chylous ascites.

British Journal of Venereal Diseases, London

29:121-196 (Sept.) 1953

Arthritis and Venereal Urethritis. D. K. Ford.—p. 123.
*Therapeutics of Non-Gonococcal Urethritis. A. H. Harkness.—p. 134.
Role of Organisms of Pleuropneumonia Group in Human Genital Infections. C. S. Nicol and D. G. F. Edward.—p. 141.
*Comprehensive Treatment for Gonococcal and Non-Gonococcal Urethritis. T. Lyall.—p. 151.
Case of Reiter's Syndrome Treated with Chloramphenicol. D. Wheatley. —p. 162.
Masked Perforation of Gastric Ulcer in Case of Tabes Dorsalis. J. O. Doyle and D. J. Campbell.—p. 164.
Simple Staining Method for Trichomonas Vaginalis. W. Fowler.—p. 166.
Potassium Iodide in Treatment of Syphilis. P. Keen.—p. 168.
An Investigation into Use of Cardiolipin Antigens: III. Choice of Formula for Cardiolipin Wassermann Antigen for Use in Whitechapel Wassermann Technique. I. N. O. Price.—p. 175.
Treatment of Gonorrhoea with Terramycin: Comparison with Penicillin. R. R. Willcox.—p. 179.

Therapeutics of Nongonococcic Urethritis.—Of 769 patients with primary nongonococcic urethritis of venereal origin, (33 of whom had Reiter's disease), 208 were treated with oxytetracycline (Terramycin), 316 with chlortetracycline (Aureomycin), 93 with chloramphenicol (Chloromycetin), 86 with streptomycin sulfate, 52 with dihydrostreptomycin, and 14 with streptomycin calcium chloride. The dosage of oxytetracycline, chlortetracycline, and chloramphenicol was 0.5 gm. every six hours for four days; when blood-borne complications were present the treatment was continued for 10 to 14 days. The dose of chloramphenicol was later increased to 0.75 gm. given every six hours, as the lower dose was frequently ineffective. The streptomycin preparations were given in a dose of 1 gm. in 4 cc. of water once daily for five days and twice daily for 10 to 14 days in cases of Reiter's disease. Chlortetracycline and oxytetracycline in particular attain a high concentration in the urine, and the therapeutic effect can be enhanced by restricting fluid intake by one-half during the treatment and by instructing the patients to pass urine frequently however small the amount. Infection with Trichomonas vaginalis was present in 59 patients and was resistant to the antibiotic treatment. Vaginal and/or cervical swabs gave positive cultures for pleuropneumonia-like organisms in 28 of 55 patients (51%); six strains were completely insensitive to dihydrostreptomycin and chlortetracycline in concentrations that would be present in the blood, moderately sensitive to streptomycin and chloramphenicol, and very sensitive to oxytetracycline in vitro. Elementary bodies of inclusion conjunctivitis type were seen in 10 patients. In patients who responded to treatment, the discharge ceased in two to four days and symptoms disappeared in one to seven days. The following cure rates were obtained: 86.5% with oxytetracycline; 63% with chlortetracycline; 39% with streptomycin; and 36% with chloramphenicol. A cure was also obtained with oxytetracycline in 24 of 31 patients who had not responded to chlortetracycline. The apparent superiority of oxytetracycline was confirmed by statistical analysis in a second series of 156 patients who were treated with the same drugs as the 769 patients but who were given these drugs at random to avoid any inclination on the part of the physician to give patients with severe disease the drug that was thought to be best, and to patients with mild disease the drug that might seem to effect a certain cure. The following cure rates were obtained: 87% with oxytetracycline; 59% with chlortetracycline; 38% with streptomycin; and 31% with chloramphenicol. Patients with Reiter's disease were best treated with fever therapy combined with oxytetracycline or chlortetracycline.

Treatment for Gonococcic and Nongonococcic Urethritis.—A search was made for a therapeutic procedure that would achieve success in the treatment of both gonococcic and nongonococcic urethritis when the circumstances were such that a microscopic diagnosis before the treatment was impracticable and at the same time would eliminate the risk of the development of a coincidentally acquired syphilis. After an extensive review of the literature on treatment of gonococcic and nongonococcic infections of the urinary tract and of syphilis with sulfonamides alone, with various antibiotics, and with sulfonamides combined with antibiotics, it was decided that every patient with urethritis, whether gonococcic or nongonococcic, should be given a course of combined streptomycin-sulfathiazole therapy. Thus 325 men with gonorrhea and 215 men with nonspecific urethritis treated in two clinics between July 1, 1952, and March 31, 1953, were given a dose of 1 gm. of streptomycin or dihydrostreptomycin by deep subcutaneous injection together with 6 gm. of sulfathiazole in four divided doses daily for five days (total dose: 5 gm. of streptomycin and 30 gm. of sulfathiazole). An average cure rate of 90% was obtained with this combined therapy in 292 patients with gonorrhea and nonspecific urethritis followed up for 12 or more days after initiation of treatment; 169 (92.8%) of 182 patients with gonorrhea and 94 (85.4%) of 110 patients with nonspecific urethritis were considered successfully treated. Urethral discharge was eliminated within 48 hours of the start of the treatment in 238 patients (96.7%) of 246 patients with gonorrhea and in 152 (92.1%) of 165 patients with nonspecific urethritis examined on the second day of the treatment. No toxic effects attributable to streptomycin were observed. Of the 540 patients treated, 394 were followed up for five days or more, and only 5 (1.26%) of the 394 showed toxic signs attributable to sulfathiazole. The injection technique required

for the administration of 1 gm. of streptomycin is no more difficult than that required for the administration of 200,000 units of penicillin in aqueous suspension. Combined treatment with streptomycin parenterally and sulfathiazole orally proved highly effective in both gonococcic and nongonococcic urethritis. Where an immediate microscopic diagnosis cannot be made, concurrent combined therapy with streptomycin and sulfathiazole is recommended for the treatment of urethritis in preference to penicillin therapy that is ineffective in the treatment of most patients with nongonococcic urethritis and carries with it the risk of masking the development of syphilis.

British Medical Journal, London

2:1389-1442 (Dec. 26) 1953

Nephrotic Syndrome. J. R. Squire.—p. 1389.
Treatment of Blood Disorders with A.C.T.H. and Cortisone. Second Report to Medical Research Council.—p. 1400.
Clinical Spectrum of Obesity. J. H. Sheldon.—p. 1402.
*Assessment and Results of Obesity. H. M. Sinclair.—p. 1404.
Maylard-Sonnenburg Method of Intestinal Anastomosis. W. J. Moore and J. Forrest-Hamilton.—p. 1407.
Penta-Erythritol Tetranitrate in Treatment of Angina. D. Weitzman.—p. 1409.
Appendicitis in Course of Measles. W. H. Galloway.—p. 1412.
Laryngeal Microphone as an Aid to Treatment of Bulbar Poliomyelitis F. D. Stott.—p. 1414.

Assessment and Results of Obesity.—Obesity, in which the amount of body fat is greater than normal, should be distinguished from overweight, in which the body weight is greater than normal in relation to such factors as race, sex, body type, height, and age. Obesity cannot be satisfactorily assessed by measuring the body weight, but it can be estimated with reasonable accuracy in adults by determining the thickness of the subcutaneous tissue, in which approximately half the amount of fat in the body is found. The problem of assessment is more complicated in children, because of the spurts in growth to which they are subject and because the distribution of subcutaneous fat varies with age. Overnourishment during growth, which leads to early maturity and a shortened life span in the lower animals, is probably harmful to children. Statistics show that at all ages men and women who are overweight have a higher mortality than the average, and the greater the degree of obesity the greater the mortality. Obesity is a forerunner of diabetes from middle life onward in fully 90% of the cases, and atherosclerosis is most frequent among the obese. Both of these conditions are characterized by an increase in the formation of cholesterol. Hereditarily obese mice in the prediabetic phase may be likened to patients with hypothalamic obesity; both have increased lipogenesis, which causes them to overeat. Diabetes characterized by a high degree of insulin resistance develops by the 9th or 10th week of life in these mice and results in their premature death. Obese mice in which insulin-resistant diabetes has appeared may be likened to the middle-aged obese diabetic; both have hypercholesterolemia. There is no relation between the serum cholesterol level and the habitual dietary intake of cholesterol, but increases in the amount of dietary fat, which tend to produce obesity, will raise the serum cholesterol and result in atherosclerosis. Weight reduction substantially lowers mortality and decreases the incidence of diseases connected with obesity among the overweight.

Dermatologica, Basel

107:369-468 (No. 6) 1953. Partial Index

Recent Studies on Alkali Neutralization and Alkali Resistance of the Skin. A. Wacek.—p. 369.
*Erythema Multiforme Exudativum; Etiological Study of Idiopathic Type. M. Ruiter and H. Hamminga.—p. 418.

Erythema Multiforme Exudativum.—In recent years internists, rather than dermatologists, emphasized the great importance of Mycobacterium tuberculosis and Streptococcus hemolyticus as causative agents for erythema multiforme exudativum. At the dermatological department of the University of Groningen, Netherlands, 17 patients with typical erythema multiforme exudativum, corresponding in every respect with the criteria established by Hebra, were studied by the authors concerning these two causative agents. Results of Pirquet's and Mantoux tests, roentgenologic examination of the lungs of the patients, otolaryngological and dental examination for local foci, and serologic examinations for streptococcic infections did not furnish any evidence in these patients of either causative agent. The authors believe this discrepancy can be explained by the fact that those workers have not sufficiently differentiated the classic forms of erythema multiforme exudativum from the somewhat similar erythema multiforme syndrome. The cutaneous manifestations of the erythema multiforme syndrome are much less characteristic than the well-defined patch or the flat papule described by Hebra as the primary florescence of a typical case of erythema multiforme exudativum. While the simultaneous occurrence of erythema nodosum has not been observed with the idiopathic type, such combined occurrence is not rare with the erythema multiforme syndrome, particularly in those cases in which internal disease processes, particularly tuberculous or streptococcic infections, and occasionally even drug hypersensitivity, form the basis for the cutaneous manifestations. While the causative factors of erythema multiforme exudativum are still unknown, the syndrome can be produced by many different causative factors. Any clinical diagnosis that is not based on morphological factors and is, therefore, not sufficiently distinctive must lead to errors. In the future the study of the causative factors must distinguish sharply between the two discussed groups on the basis of clinical morphology.

Deutsche medizinische Wochenschrift, Stuttgart

79:1-68 (Jan. 1) 1954. Partial Index

Pathogenesis and Diet in Hypertension. H. Staub.—p. 1.
Myocardial Infarction. F. Lange.—p. 6.
Primary Pulmonary Right Cardiac Insufficiency. K. Spang.—p. 9.
Incidence of Cardiac Diseases in Course of Rheumatic Fever and Drug Treatment of Rheumatic Carditis and Related Diseases. F. Grosse-Brockhoff.—p. 15.
*Recent Developments in Treatment of Initial Phase of Apoplectic Attack with Aminophylline Preparation (Euphylline). F. Mainzer.—p. 37.
*Treatment of Arterial Hypertension with Rauwolfia Alkaloid Reserpin (Serpasil). G. Damm and H. Trautner.—p. 39.

Aminophylline in Initial Phase of Apoplectic Attack.—In 1949, Mainzer introduced the treatment of the initial phase of an apoplectic attack with intravenous injections of aminophylline (Euphyllin). Up to December, 1952, 126 cases were reported by the author in previous papers and by other workers in which favorable results were obtained with this method. It appeared that the aminophylline is fully effective only when given in the ischemia-conditioned functional phase of the apoplectic attack. The percentage of success is reduced from 80 to 45% in patients to whom the drug was given within five hours after the attack. The drug should be given within one hour of the initial attack, although coma and mild paresis disappeared in two of the author's patients who were given the drug within eight hours, and one patient responded favorably to the drug given 11 hours after the initial attack. The author emphasizes the importance of the intravenous route of administration, while oral, rectal, or intramuscular administration may result in failure. Refractory headache in patients with cerebral arteriosclerosis and hypertension (one illustrative case in an 84-year-old woman is described) responded favorably to aminophylline. Increased permeability of the blood-cerebrospinal fluid barrier produced by the aminophylline is probably responsible for its effect in the initial stage of the apoplectic attack.

Rauwolfia Alkaloid Reserpine in Hypertension.—Reserpine (Serpasil), was given a therapeutic trial in 44 patients with arterial hypertension in whom the high blood pressure had not been significantly reduced by bed rest, salt-free diet, and occasional digitalization for three to six weeks. These measures were continued after the institution of reserpine. The drug was given in three tablets, each of 0.5 mg., daily; three patients received up to six tablets daily. Marked lowering of the blood pressure was obtained in 20 of the 44 patients with all types of arterial hypertension. The drop in blood pressure exceeded that obtained with the usual hospital procedures. Parallel improvement of subjective complaints was observed in many patients. Lowering of blood pressure resulted from combined treatment with reserpine and Nepresol (1, 4-dihydrazinophthalazine) in nine patients who did not respond to reserpine alone. A condition

similar to collapse resulted from the pronounced reduction of blood pressure in two patients with severe arteriosclerotic changes. It seems that reserpine reduces the blood pressure by exerting an effect on the central nervous system. Lowering of blood pressure was likewise obtained in some patients with phenobarbital, bed rest and salt-free diet. Phenobarbital also acts as a depressor agent on the central nervous system.

East African Medical Journal, Nairobi

30:399-446 (Oct.) 1953. **Partial Index**

Anthrax Meningo-Encephalitis. A. B. Raper.—p. 399.
*Toxicity of Chloroquine in Hepatic Amoebiasis in Africans. J. Wilkinson. —p. 403.
Examination of Red-Blood Counts, Haemoglobin, Packed Cell Volume, Serum Iron and Iron Continuing Capacity of Blood and Serum of Small Series of Pregnant Women at Mulago Hospital. E. G. Holmes. —p. 427.

Toxicity of Chloroquine in Hepatic Amebiasis.—Chloroquine sulfate was used in the treatment of 112 Bantu Africans with hepatic amebiasis. Toxic reactions were noted in 52, and were severe in 10. The toxic effects were unpleasant, but not fatal, and they were directly related to the dosage of the drug and to the length of treatment. Women had severer toxic reactions than men. Vomiting sometimes required that use of the drug be stopped. The toxic effects were reversible, passed off within 48 hours after medication had been discontinued, and caused no permanent damage. Chloroquine may have a temporary toxic effect on the nervous system. It is assumed that the greater incidence of toxic reactions in these patients was caused by their poor physical condition. They were smaller in stature and lighter in weight than Europeans, also they were malnourished. Chloroquine should be used with caution in persons in whom blurring of vision and giddiness may have dangerous results, such as in drivers and pilots. These should not be allowed to continue their work while being treated with chloroquine, at least in the doses required in amebiasis.

Edinburgh Medical Journal

60:461-494 (Dec.) 1953

Cancer of the Larynx. I. S. Hall and J. F. O. Mitchell.—p. 461.
Renal Sclerosis, "Post-Radiation Nephritis" Following upon Irradiation of Upper Abdomen. H. Russell.—p. 474.

Electroencephalography & Clin. Neurophys., Montreal

5:1-162 (Feb.) 1953. **Partial Index**

Analysis of Central Responses to Photic Stimulation in Normal Adults. A. C. Mundy-Castle.—p. 1.
EEG and Reaction to Photic Stimulation as Index of Anxiety-Proneness. G. A. Ulett, G. Gleser, G. Winokur and A. Lawler.—p. 23.
Occurrence of Sharp Waves, Spikes and Fast Activity in Supratentorial Tumours. L. Kirstein.—p. 33.
Electroencephalographic Sequelae of Spontaneous Subarachnoid Haemorrhage. J. N. Walton.—p. 41.
Electroencephalographic Studies of Encephalopathies: II. Serial Studies in Tuberculous Meningitis. R. C. Turrell, W. Shaw, R. P. Schmidt and others.—p. 53.
*Value of Electroencephalography for Differential Diagnosis of Neurosis and Organic Brain Disease. H. C. Stehle.—p. 65.
Spontaneous Excitation of Visual Cortex and Association Areas—Lambda Waves. C. C. Evans.—p. 69.
Electroencephalographic Changes in Siblings with Hypocalcemia Due to Hypoparathyroidism. C. Hamsted and S. Brandt.—p. 101.
Simple Electronic Stimulator Suitable for Peripheral Nerve and Skin Sensory Testing. G. H. Bishop.—p. 105.
New Multilead Electrode for Intracerebral Electrography in Man. R. von Baumgarten.—p. 107.

Electroencephalography in Differentiation of Neuroses and Organic Brain Disease.—Stehle feels that because some types of organic brain damage and certain types of epilepsy are symptomatically difficult to distinguish from neurosis, it should be advantageous to obtain electroencephalographic studies on all neurotics. However, since such mass electroencephalography is not practical, patients should be screened by the physician or the electroencephalographer so that recordings are made only in cases in which there is a reasonable expectation of significant electroencephalographic findings. For some years this policy has been carried out at the author's clinic. The present study was undertaken to assess the value of electroencephalography as an aid to the diagnosis of neurosis using currently accepted criteria for referral. Answers were sought to the following questions: 1. What is the type and incidence of electroencephalographic abnormality in patients with a presumed diagnosis of neurosis who are referred for electroencephalographic examination? 2. With what "neurotic" symptoms do electroencephalographic abnormalities correlate? The 263 veteran patients studied by electroencephalography were highly selected, not representative of all neurotics, but only those who (though presumably neurotic) were suspected of having organic brain disorder. Awake and sleep electroencephalograms were obtained in 200 cases and only awake records were obtained in 63 cases. Electroencephalograms made during sleep in 200 patients revealed significant abnormalities with clinical correlation in 19%, seizure discharges accounting for 14%. In the group examined only when awake, the incidence of significant abnormalities with clinical correlation was much lower. Epileptic seizure discharges were never recorded in the electroencephalogram made while awake but only during the drowsy or sleep state. This seems to indicate the advantage of making studies during sleep. The author feels that the following generalization is permissible; the less severe the abnormality the more essential is electroencephalographic study during sleep. The diagnostic use of electroencephalographic study appears indicated in neurotic patients with symptoms such as major convulsions, abdominal complaints, amnesia, visual disturbances, rage attacks, nausea and vomiting, and weakness-fainting-blackouts. This appears especially true if the clinical history gives any indication of a previous head injury or cerebral infection. It was found that 27.5% of those showing normal electroencephalographic records during sleep gave a history of cerebral trauma or infection, but 45% of those showing abnormal electroencephalographic findings gave such a history. In the groups with psychomotor, thalamic, and hypothalamic epilepsy 50% had a history of head injury.

Glasgow Medical Journal

34:481-518 (Nov.) 1953

Observations on Haemophilia and "Christmas Disease." A. H. Imrie and A. J. V. Cameron.—p. 481.
Rigidity of Old Age: Clinical and Pharmacological Study. J. C. Brocklehurst.—p. 486.
*Functional Uterine Haemorrhage in Puberty and Adolescence. A. M. Sutherland.—p. 496.

Uterine Hemorrhage in Puberty and Adolescence.—Sutherland made studies on 200 girls, 20 years of age or younger, who had abnormal uterine bleeding. Of these, 179 girls were between 16 and 20 years of age. Sixteen of the 200 had been pregnant, 9 having had one delivery at full term and the other 7 having had one abortion. He is of the opinion that it is impossible to make a diagnosis of functional uterine bleeding until the endometrium has been examined histologically. Dilatation and curettage was done in all but one of the 200 girls. In the patient not subjected to curettage uterine fibroids of about the size of a four month pregnancy were present and hysterectomy was necessary. In those with a reasonably regular cycle curettage was done during the premenstrual phase. Tuberculosis of the endometrium was found in 8 cases and chronic endometritis in 10 cases. Cystic glandular hyperplasia was found in only 31 cases and endometrial atrophy in 4 cases. In the remaining 147 cases the endometrium appeared normal. An attempt was made to follow-up as many as possible of the last 50 patients. They were asked to report in two months, and subsequent attendances varied with the individual case. Two patients with endometrial tuberculosis were excluded from this follow-up. They were treated with streptomycin and *p*-aminosalicylic acid. Of the remaining 48 cases 5 could not be traced. In 20 of 43 patients, menstruation remained normal after dilatation and curettage alone during an average follow-up period of 11 months. In six others menstruation returned to normal after curettage, but abnormal bleeding recurred after an average period of six months. There was no relationship between the endometrial histology and the results of curettage. In three of six cases of endometrial hyperplasia, the curettage had no effect, and in the three others it was followed by a return to a normal cycle. The antimenorrhagic factor, which is a non-

saponifiable fraction of mammalian liver tissue suspended in a neutral base, was used in 19 of the remaining 23 patients. Cure was obtained in 12, but in 6 of these a second course of the drug had to be given. One other patient failed to return, and in the remaining six patients the antimenorrhagic factor was ineffective. Treatment with estrogen preparations proved effective in four of the six cases in which it was tried.

Journal of Bone and Joint Surgery, London

35-B:517-722 (Nov.) 1953

Rest and Movement. G. Perkins.—p. 521.
Early Treatment of Paraplegia from Fractures of Thoraco-Lumbar Spine. F. W. Holdsworth and A. Hardy.—p. 540.
Tuberculosis of Elbow: Study of 31 Cases. J. N. Wilson.—p. 551.
Preparation and Use of Heterogenous Bone Grafts. M. Guilleminet, P. Stagnara and T. Dubost-Perret.—p. 561.
Development of Congenital Dislocation of Hip. E. W. Somerville.—p. 568.
*Tuberculosis Transmitted by Banked Bone. J. I. P. James.—p. 578.
Congenital Kyphosis. A. C. Bingold.—p. 578.
*Mesenchymoma (Angioliposarcoma) of Thigh. M. Pemberton and P. I. Cromack.—p. 584.
Parosteal Lipoma. H. A. T. Fairbank.—p. 589.
Case of Tuberculosis of Ischium. G. S. Tupman.—p. 590.
Parathyroid Osteodystrophy: Report of Case. H. J. Richards.—p. 593.
Osteoarthritis of Hip: Study of Nature and Evolution of Disease. M. H. M. Harrison, F. Schajowicz and J. Trueta.—p. 598.
Role of Capsular Changes in Osteoarthritis of Hip Joint. G. C. Lloyd-Roberts.—p. 627.
Bone Cysts of Osteoarthritis. J. W. Landells.—p. 643.
Recovery in Partly Paralysed Muscles. D. D. B. Morris.—p. 650.

Tuberculosis Transmitted by Banked Bone.—James says that during the last year ribs from thoracoplasties performed in cases of pulmonary tuberculosis have been stored in the bone bank. The ribs have been frozen at -15 to -20 C in a solution containing penicillin and streptomycin. They have been used in a wide variety of orthopedic procedures. In the last months four tuberculous abscesses have been discovered. After removal of the infected bone chips and curettage, the wounds healed without further incident; it is possible that further manifestations may appear. This unfortunate sequel makes it obvious that such material is unsuitable for bone grafting. The four cases are briefly described.

Mesenchymoma (Angioliposarcoma) of Thigh.—According to Pemberton and Cromack the term mesenchymoma was first applied by Gilmour in 1943 to an unusual recurring tumor composed of embryonic connective tissue. This tumor appeared histologically benign, but recurrences developed after every attempt at local removal. Since then, three more cases of a similar character have been described by Symmers and Nangle (1951), who also recorded the fatal termination of Gilmour's case. Pemberton and Cromack present two cases of mesenchymoma occurring in the thigh of two women, aged 47 and 50, respectively. In the first patient the limb was removed by disarticulation at the hip joint, all the muscles being divided as high as possible. One year after the operation the patient was in good health and there was no sign of recurrence. The tumor was ovoid and soft and measured 19 by 15 cm. It was partly surrounded by a thin diaphanous capsule. On gross section the tumor was separated into lobules by fibrous septa. It encircled the femur for almost three-quarters of its circumference, extended upwards from the knee joint to the uppermost limits of the vastus intermedius. Although it was mainly confined to the quadriceps group, it involved also the insertion of the adductor muscles into the shaft of the femur. Microscopic examination disclosed angioblastic, fibroblastic, and lipoblastic tissues. In the second patient an extensive local excision had been done at another hospital. When one year later a recurrence was observed, it was removed together with the fascia of the surrounding muscles. There was a further recurrence one year later; local excision was again undertaken. There was yet another local recurrence nine months later, again treated by excision. At the fourth operation it was obvious that the tumor was infiltrating the surrounding tissues. Amputation was, therefore, undertaken four years after the onset of symptoms and three years after the first local excision. One year after amputation there was no evidence of a return of the lesion. The term mesenchymoma is appropriate, because of the resemblance of the primitive fibroblastic component of the tumor to mesenchymal tissue. Confusion in terminology may arise, however, because the term mesenchymoma has also been applied to other tumors such as myxomas, mixed malignant tumors of mesodermal origin, and even to undifferentiated sarcomas. The two cases presented conform to the pattern of Gilmour's case to which he applied the description mesenchymoma. Since recurrence seems invariably to follow local removal a relatively drastic surgical policy is justifiable and indeed necessary.

Journal of Laryngology and Otology, London

67:701-778 (Dec.) 1953

Vestibular and Acoustic Symptoms and Other Phenomena Following Treatment with Streptomycin and Dihydrostreptomycin. H. C. Andersen, H. H. Ohrt and others.—p. 701.
Elastic Tissue in Middle-Ear Cavity. M. Harty.—p. 722.
Blown Plastic Inlays in Mastoid Surgery. D. A. Draffin.—p. 730.
Testing for Otosclerosis, and Description of Quantitative Gellé Test. D. MacFarlan.—p. 740.
Pathological Changes in Blood-Vessels and Nerves of Oesophagus Due to Corrosion. B. Gušić.—p. 745.

Lancet, London

2:1323-1366 (Dec. 26) 1953

Education for Elizabethans. Lord Radcliffe.—p. 1323.
Investigation of Obliterative Arterial Disease of Lower Limb. D. Messent, R. E. Steiner and J. F. Goodwin.—p. 1324.
Reflections on Diabetes Mellitus: Answers to Questionary. G. F. Walker.—p. 1329.
*Intravenous Procaine in Treatment of Chest-Wall Injuries. P. G. Bevan.—p. 1332.
Two Cases of Myxoedema Attributed to Iodide Administration. M. E. Morgans and W. R. Trotter.—p. 1335.
Alopecia, Optic Atrophy, and Peripheral Neuritis of Probably Toxic Origin. W. J. C. Symonds.—p. 1338.
Infants' Legs and Sleeping Position. M. P. Pick.—p. 1339.
Mycetoma of Testis. P. R. R. Clarke.—p. 1341.

Intravenous Use of Procaine in Chest Wall Injuries.—Bevan tried procaine for the relief of pain associated with chest wall injuries. The injury was often minor, though some cases of major trauma were also included. The pain was of three types—localized to the site of injury, diffusely spread over a wide area of the chest, or referred forward along the intercostal nerves. Coughing, inspiration, and movement of the trunk or upper limb were often much restricted. The procaine was injected into a vein of the forearm from a 20 ml. syringe. After various modifications, the standard dose adopted for a normal adult was 20 ml. of 1% procaine, with adjustment for age and physique. At first the injections were given slowly over 15 minutes; but later the time was reduced to 5 minutes. Each injection was given with the patient recumbent, to counteract the vasodilator effect of procaine. He then rested half an hour and was sent home by ambulance. He was seen again next day and again a few days later. The procaine was prepared in sealed 20 ml. ampuls and contained no epinephrine (Adrenaline) or bacteriostatic. Convulsions, which may lead to respiratory paralysis, and peripheral vascular failure resulting from medullary depression are dangers in procaine therapy, which are probably due to idiosyncrasy. To safeguard the patients against these threats a tray was at hand containing thiopental (Thiopentone) sodium to antagonize the convulsant action of procaine and ephedrine to combat the medullary depression. However, in giving 82 injections there was no need to use these drugs. In three cases the patient lost consciousness and in two speech was lost, but these effects were transient. In two elderly men the pulse was irregular for an hour. Pain was relieved in the large majority of the patients, and their respiratory function and general condition improved. The relief was greater in fractures than in soft tissue injury, and the benefit was greatest when the injection was given soon after the injury. The relief was immediate or was delayed for a few hours. It lasted for about 20 hours. A second injection was never so effective as the first. Estimation of procaine levels in blood and urine confirmed that procaine is excreted from the blood within an hour. Its effect over hours or days is probably not due to continuing anesthetic action but to a breaking of the vicious circle of pain to muscle spasm to pain. In control cases, intravenous injections of isotonic sodium chloride solution were of some benefit, but it was much less than with procaine.

Minerva Ginecologica, Turin

5:589-628 (Sept. 30) 1953

New Methods for Staining Spermatozoa and Determining Live and Dead Cells. T. Bonadonna.—p. 589.
Infusion of Concentrated Suspension of Red Blood Cells in Obstetrics and Gynecology. L. Milani and W. Torsiglieri.—p. 595.
*Vitamin K_1 as Antidote to Drugs with Dicumarol-Like Effect: Clinical Observations. L. Gagliardi.—p. 598.
Experimental Studies of Corticotropin Action on Genital Apparatus of Rats (Mus Decumanus) After Removal of Ovaries. P. Quaini and C. Durando.—p. 606.
Gonadotropins in Cerebrospinal Fluid. E. Revelli.—p. 611.
Use of Quaternary Bases of Ammonium as Antiseptics in Obstetrics and Gynecology. A. Massano and S. Monticelli.—p. 618.

Vitamin K_1 and Anticoagulant Therapy.—Synthetic vitamin K_1 (Konakion) was administered to four patients receiving Marcoumar (3[1'-phenyl-propyl]4-hydroxycoumarin) and three receiving ethyl biscoumacetate (Tromexan ethyl acetate). The good effects of the vitamin, which was given orally in doses of 10 to 12 mg., became evident in most instances six hours after its administration. In one patient receiving Marcoumar, the prothrombin time was shortened from 70 to 32 seconds in 10 hours and from 32 to 28 seconds in the next 24 hours. Twelve hours after the administration of a second 10 mg. dose of the vitamin, it was reduced to 23 seconds. Equally good effects were obtained in the other six patients. Similar results have been reported by American and English authors. They, however, had given their patients a single dose of 500 mg. of the vitamin, whereas Gagliardi never used more than from 10 to 24 mg. of it. Synthetic vitamin K_1 greatly improves the prognosis of patients receiving anticoagulants with a bishydroxycoumarin-like action, because with its administration the hemorrhagic complications secondary to this therapy can be prevented.

Minerva Medica, Turin

44:1421-1456 (Nov. 14) 1953

Disorders of Intraventricular Conduction During Total Arrhythmia. V. Nazzi and A. Anselmino.—p. 1421.
Pneumographic Investigation of Mediastinum. E. Valentino.—p. 1431.
*Cortisone in Treatment of Tetanus. C. Cossali and A. Biella.—p. 1435.
Aromatic Derivative of Mustard Gas in Treatment of Tuberculous Lymphadenitis: Preliminary Note. L. Costelli.—p. 1438.

Cortisone Therapy in Tetanus.—Three patients with tetanus were given cortisone as supplement to antitoxin serum and penicillin. Although the authors could not judge what part the hormone played in attenuating the course of the disease, they say that it did have a favorable effect on the clinical course and brought relief to the patients by rapidly lowering the elevated temperature, reducing the intensity and number of spasms, and improving the patients' vital sense and appetite. The first patient, a 5-year-old girl, was hospitalized with a diagnosis of meningitis. After the presence of tetanus was established, she was given antitoxin serum, penicillin, and cortisone. She received the hormone first in two 50 mg. injections and later in one 50 mg. injection daily for a total dose of 1,500 mg. She recovered in about two weeks. Antitoxin serum and penicillin were given to the second patient, a 17-year-old girl, but her already severe condition was aggravated by the therapy. Cortisone was then administered in doses of 200 mg. for one day, 100 mg. for 6 days, and 50 mg. for 14 days. There was a noticeable improvement with disappearance of the spasms and recovery. The severe infection had remained undiagnosed in the third patient, a 64-year-old man. After the correct diagnosis was established, massive therapy was instituted, and, in addition to antitoxin serum and penicillin, he was given 300 mg. of cortisone for three days, 200 mg. for seven days, 100 mg. for seven days, and 50 mg. for three days. This patient also recovered. The cortisone action was particularly evident in this case because patients in similar conditions who had been treated at the same hospital without cortisone died. According to the authors, this hormone potentiates the organism's means of defense and prevents their exhaustion thus enabling the specific defense systems to intervene and bring about the recovery. A definitive evaluation of the value of cortisone in the treatment of tetanus is, however, still premature.

Nordisk Medicin, Stockholm

50:1493-1526 (Oct. 29) 1953. **Partial Index**

Athletic Heart. T. Sjöstrand.—p. 1493.
Danger in Use of Morphine in Cor Pulmonale. S. Samúelsson.—p. 1496.
*Development of Low Salt Syndrome During Dehydration in Patients with Cardiac Decompensation. B. Harvald and P. Astrup.—p. 1499.
Etiology of Cerebral Palsy. O. Scheel Thomsen.—p. 1504.
Incidence of Bacterial Contamination in Vials Used in Hospital. O. Kylin and H. Ekstrand.—p. 1507.

Development of Low Salt Syndrome During Dehydration in Cardiac Insufficiency.—Harvald and Astrup discuss the danger of the development of secondary uremia in patients with cardiac insufficiency treated with mercurial diuretics and salt poor diet. They followed 16 patients during dehydration treatment consisting of one or two mersalyl injections (1-2 ml.) weekly in connection with a salt poor diet containing only a small amount of salt daily. Powerful dehydration therapy with salt poor diet can gradually lead to the so-called low salt syndrome. The subjective symptoms of salt depletion are fatigue, apathy, thirst, anorexia, general muscular weakness, muscular spasms, and tendency toward syncope. The syndrome is accompanied by reduced renal function and abnormally high blood urea concentration, usually also hypochloremia, and possibly hypopotassemia and hyponatremia. On the development of the low salt syndrome the mersalyl medication must be stopped at once and sodium chloride given by the mouth, whereupon the symptoms disappear. When the blood urea and serum electrolyte concentrations are returned to normal the mersalyl treatment can be resumed. The diuretic effect of mersalyl is more dependent on the serum chloride concentration than on the serum sodium concentration. It is suggested that mersalyl affects the chloride resorption in the renal tubules, not, as usually assumed, the tubular resorption of sodium.

50:1595-1628 (Nov. 19) 1953. **Partial Index**

*Anemia and Sideropenia After Gastrectomy. S. Wallensten.—p. 1601.
Cortisone, Ascorbic Acid and Amyloid Formation: Clinical Considerations on Teilum's Views Concerning Relation Between Them. G.-A. Johansson.—p. 1605.
*Lymphosarcomas of Stomach. J. Engberg.—p. 1610.
Acute Abdominal Disorders Due to Meckel's Diverticulum. E. Christensen.—p. 1614.
Perforated Duodenal Ulcer and Acute Abdominal Infections During Corticotropin/Cortisone Treatment. K. Hatteland.—p. 1616.

Anemia and Sideropenia After Gastrectomy.—Wallensten's 364 cases of gastroduodenal ulcer operated on (Billroth 2) at the Serafimerlasarett consisted of 281 cases of duodenal ulcer in 201 men and 80 women, of whom 205 were under 50, and 83 cases of gastric ulcer in 58 men and 25 women, of whom 35 were under 50. On follow-up examinations with reference to postoperative anemia Vahlquist's values of 13.6 gm. per 100 cc. for men and 11.5 gm. per 100 cc. for women were taken as normal. Postoperative hypochromatic anemia was found to be common after Billroth 2. Pernicious anemia did not occur. There was a high percentage of sideropenia in these anemias. The longer the time after operation, the greater was the frequency of both anemia and sideropenia. The anemia was seen oftener in women operated on before the menopause than after it. According to earlier investigations anemia is infrequent in gastric and duodenal ulcers operated on according to Billroth 1. Billroth 1 is advocated as the method of treatment for gastric ulcer and duodenal ulcer in women, but in duodenal ulcer in men Billroth 2 should continue to be the procedure, since in these cases the danger of recurrence is greater after Billroth 1. The cases should be closely followed postoperatively with regular blood control and anemia compensated by iron intravenously as oral therapy is ineffective.

Lymphosarcoma of Stomach.—From 1 to 2% of the malignant tumors of the stomach are represented by mesenchymal tumors, fully half of which are lymphosarcomas. About 600 cases of these lymphosarcomas have now been published. Recent statistics, Engberg states, show that the five year survival rate after radical operation for lymphosarcoma of the stomach is high and increasing. The importance of diagnosis is stressed. It may be extremely difficult. To date correct preoperative diagnosis has been made in only a few cases, but with the pos-

sibility of lymphosarcoma of the stomach in mind in "atypical carcinomas" diagnosis should be possible much earlier. The tumors manifest themselves at any age but occur mainly at an earlier age than carcinomas and are somewhat more frequent in men than in women. The symptoms are vague and the course is slow. In fully 50% a large palpable tumor is found or at least a definite resistance in the epigastrium is felt on the first consultation. The absence of demonstrable ascites and metastases should suggest sarcoma. In a relatively young patient in good condition with a large epigastric tumor, demonstrable roentgenologic changes in the stomach, and long history, the diagnosis of lymphosarcoma should be considered. Benzidine tests in the feces are less constantly positive and achylia is less frequent than in carcinoma. While roentgen examination is a valuable aid there are no pathognomonic signs. So far, diagnosis by means of gastroscopy has been the exception. Treatment is total or subtotal resection of the stomach and resection of omentum, most often with intensive roentgen treatment. Lymphosarcomas are exceedingly radiosensitive. Laparotomy is contraindicated only in the most advanced cases in cachetic patients. The excellent results now reported depend on intrepid radical intervention. Even advanced cases of so-called "inoperable carcinomas" call for exploratory laparotomy to rule out possible lymphosarcomas. Two cases recently treated in Randers Centralsykehus are described.

Semaine des Hôpitaux de Paris

29:3889-3954 (Dec. 6) 1953

New Data on Metabolism of Hemoglobin. H. Benard, A. Gajdos and M. Gajdos-Torok.—p. 3889.
*Psychosurgery for Intractable Pain: Based on 57 Cases. D. Petit-Dutaillis, R. Messimy and Mrs. L. Berges.—p. 3893.
Research on Individualization of Staphylococci: Application to Study of Staphylococcic Endemia. R. Turpin, J. Pillet, J. Calmels and Mrs. J. Pillet.—p. 3904.
Value of Phosphorus 32 in Treatment of Malignant Giant Follicular Lymphoma. H. Desgrez, R. A. Guérin, Mrs. M.-T. Guérin and F. Heitz.—p. 3910.

Psychosurgery for Intractable Pain.—It is emphasized that psychosurgery is definitely a last resort in cases of intractable pain. Prefrontal lobotomy, the technique most frequently used, can ameliorate pain in some cases, but rarely suppresses it definitively. Of this series of 57 patients, 43 were operated on for severe pain from neoplasms and/or metastases, 4 for cenesthopathia, 3 for sympathalgia, 2 for pain resulting from trauma to the brachial plexus, 1 for pain from a thalamic syndrome, 1 for pain from the stump of an amputation for osteomyelitis, 1 for causalgia from anesthetic leprosy, 1 for causalgia with edema following a fracture that was treated by means of a cast, and 1 for tabes with cystalgia. The authors have drawn the following conclusions. 1. Lobotomy done bilaterally in one operation, which was performed in 23 of the patients in this series, often yields good results. There are, however, serious disturbances in personality accompanying the relief of pain. There were three deaths attributable to intervention in this group. 2. Bilateral lobotomy in two stages, done in seven patients, did not yield as constant results in relieving pain. 3. Unilateral lobotomy, which was done 5 times on the right and 15 times on the left, often has a favorable result; but the effect is not so long-lasting as in the bilateral operation and it is often difficult to get the patient's family to accept another operation. The personality disorders often associated with this procedure were less severe and did not last so long as those following bilateral lobotomy. It was not possible to demonstrate a dominating analgesic effect, either homolaterally or contralaterally. 4. Excision of certain areas, or topectomy, was performed in seven cases; the results were only transitory, and the procedure was believed to have contributed to death in two aged, hypertensive patients. 5. In four of the patients in this series, lobotomy was done chemically rather than surgically. It should be done bilaterally, the authors feel, and they think it is of value for delicate patients. Studies ought to be made of this procedure's particular influence on personality. 6. It is believed that the analgesic effect caused by psychosurgery is due to its effect on the autonomic nervous system by means of a thalamohypothalamic disengagement. This would explain both the sensomotor and the psychoaffective changes that occur. After lobotomy, pain is better tolerated because the autonomic nervous system has been brought down to a more primitive level, a level approximating that of children in whom, it is pointed out, the evolution of a cancer involves little or no pain. In three of the authors' patients, edema was reduced by psychosurgery, another fact in favor of the theory of a neurovegetative mechanism. This theory also accounts for the unusually good results obtained in the cases of sympathalgia and causalgia.

29:4171-4236 (Dec. 26) 1953. **Partial Index**

*Artificial Hibernation in Coronary Occlusion and Cardiovascular Surgery: Experimental Studies and Clinical Perspectives. C. Lian.—p. 4171.
*Pharmacoclinical Studies of Treatment with Acetyl-Digitoxin of Severe Forms of Asystolia and Rhythm Disturbances. Loeffler.—p. 4174.
Anticoagulant Medication in Myocardial Infarction and Angina Pectoris. M. Mouquin.—p. 4178.
Periarteritis Nodosa and Related Syndromes or "Disseminated Allergic Angiitis." J. Roskam.—p. 4184.
Neurobiological Aspect of Several Large Vascular Syndromes. H. Laborit. —p. 4190.

Artificial Hibernation.—The experiments on dogs made by the author and his associates have led him to propose artificial hibernation for the treatment of three conditions in man —myocardial infarction with shock, myocardial infarction without shock, and angina pectoris of coronary origin. It was found that ordinary anesthesia could not prevent the death of dogs in which the coronary arteries were ligated, but a dog in an induced state of hibernation could survive this procedure. The author proposes to induce a state of hibernation in patients with myocardial infarcts who are in shock. Both pharmacodynamic agents, such as Largactil (dimethylaminopropyl-N-chlorophenothiazine hydrochloride), Phenergan (10-[2-dimethylamino-1-propyl]phenothiazine), and meperidine (Dolosal), and ice bags placed on the axillas and the groin would be used. This treatment would be both preventive and curative; it should preferably be done at the first sign of infarction. The dangers resulting from the hypotension of shock would be obviated by the reduction of the metabolic needs of the body as a whole. Pain would be suppressed, cardiac irritability diminished, and the cardiac rhythm slowed. The technique of therapy proposed for human beings is described in detail. The administration of vitamins and heparin is included in the treatment, which is to last four days. In the case of myocardial infarction without shock, only pharmacodynamic agents are used, along with heparin and etaphylline. A small number of patients has already been treated in this manner; the procedure had the great advantage of stopping immediately intense pain that had not yielded to morphine. Two of the drugs used in intravenous infusion or intramuscularly to induce complete or partial hibernation in the patients with infarcts, Largactil and Phenergan, could also be used in small daily oral doses for the prevention of angina attacks. Their sympathicolytic action could presumably moderate the apparition or accentuation of coronary sclerosis and coronary spasm. In cardiovascular surgery, artificial hibernation can perform the same services that it does in general surgery. It permits of long periods of circulatory arrest without ventricular fibrillation and the loss of cerebral electric activity that ensues is purely temporary. It would seem, therefore, that the artificial heart and lung could either be dispensed with entirely or used in conjunction with artificial hibernation with hypothermia.

Acetyl-Digitoxin in Cardiac Dysrhythmia.—Acetyl-digitoxin was administered to 120 patients with severe cardiac insufficiency. Two-thirds of these patients were over 60 years old, and three-fourths of them presented one or several serious complications—thrombosis, pulmonary infarct, pneumonia, apoplexy, or Laennec's cirrhosis. Treatment with acetyl-digitoxin brought good results, particularly in all tachycardiac forms of cardiac insufficiency. The drug also proved valuable in bradycardiac forms, but it appears that less vagotropic agents would be preferable. Dosage of acetyl-digitoxin must be separately determined for each patient; rapid or slow saturations can be effected in more or less urgent cases. The drug is well tolerated. In the series reported, 43% of the patients had excellent results, 38% good results, and 19% no improvement. Other cardiotonics also gave poor results in this last group. The properties of acetyl-digitoxin place it pharmacologically between digitoxin and the glucosides of lanatose digitalis.

BOOK REVIEWS

Injuries of the Spinal Cord. Edited by George C. Prather, M.D., F.A.C.S., and Frank H. Mayfield, M.D., F.A.C.S. Cloth. $8.75. Pp. 396, with illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1953.

This monograph is written by a group of physicians who were responsible for the care of patients with injuries to the spinal cord in United States Army general hospitals during World War II. Patients with severe injuries are encountered occasionally in civilian practice, but it is only under unusual conditions such as war that a large number of cases are available for definitive study and prolonged therapy. The monograph was written to give the student and physician all of the information necessary for the diagnosis and management of these patients. The authors are all eminent specialists in their various fields. There is a brief chapter on history followed by discussions of the management of the patient in the acute and subacute phases, the neurosurgical and urological aspects, the problems of nutrition, associated injuries, the treatment of decubitus ulcers, and the management of the patient after the acute phase, with particular emphasis on rehabilitation. The authors have relied mainly on their own clinical experience in their presentation of the subject, but a fairly extensive list of references is appended to each chapter. The material is well presented. Indications for diagnostic procedure and therapeutic measures are clearly outlined. The printing is excellent and easily read. The figures are pertinent and clearly reproduced. There is an adequate index. The book should be of great value to students and physicians who are responsible for the care of these patients, particularly neurologists, neurosurgeons, orthopedists, and physiatrists.

The Radiology of Bones and Joints: An Introduction to the Study of Tumours and Other Diseases of Bone. By James F. Brailsford, M.D., Ph.D., F.R.C.P., Consulting Radiologist to Queen Elizabeth Hospital, Birmingham, England. Fifth edition. Cloth. $19. Pp. 875, with over 725 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1953.

This book is now in the fifth edition and is widely used by many radiologists in this country. Over the years the clinicians, the orthopedists, and the radiologists have had great difficulty in interpreting bone and joint disease as portrayed on the roentgenogram. With experience it is possible to make presumptive roentgenologic diagnoses that will include several conditions; for instance, the roentgenographic evidence of osteomyelitis may simulate the findings of a Ewing tumor. Often it is impossible to differentiate between the two by the roentgenologic examination alone, and it is only after correlating the roentgenologic with all the other findings, including the pathological findings, that a correct diagnosis is made and proper treatment is instituted. The author, because of his vast experience, has collected roentgenograms of many conditions, and a short discussion of these conditions is included in the text.

It is difficult to reconcile the following statement of the author with good medical practice: "The author acknowledges the great debt that we owe to clinicians and pathologists. . . . By correlation of the clinical with the histopathological and with radiography we have learned a great deal about the significance of radiographic appearances. . . . Why . . . submit the tuberculous and multitudinous other conditions to biopsy with all its risks in an attempt to secure 'confirmation'? The summations of reports from many sources on relatively limited knowledge do not add up to accuracy. The superficial resemblances of clinical, microscopical and radiological features of widely different lesions do not permit a sound teaching by 'atlas' methods." There are bone conditions in which a correct diagnosis can be made from the roentgenogram, but there are many other important lesions in which every possible aid is needed, including microscopic studies of excised tissues.

The author's descriptions are largely radiological; little consideration is given to the lesions producing the radiological appearances, and there are certain subjects, such as meningiomas, that receive only meager comment. On the other hand, this book includes a wealth of information, which should be helpful to the radiologist. The format of the book is good. The type is easy to read, but the illustrations unfortunately are mostly positive roentgenograms. The author is to be congratulated on the index. It is refreshing to be able to turn to an index of a medical book and find the subject sought.

What You Should Know About Mental Illness. By Orin Ross Yost, M.D., Medical Director, Edgewood Sanitarium Foundation, Orangeburg, S. C. With foreword by Nolan D. C. Lewis, M.D., introduction by Leland E. Hinsie, M.D., and note by Norman Vincent Peale, D.D. Cloth. $3.50. Pp. 165. Exposition Press Inc., 386 Fourth Ave., New York 16, 1953.

Dr. Yost has written a most readable little book. Although he is a prominent figure in the field of psychiatry, he has by no means written this book specifically for psychiatric consumption. In considering the Baconian tripartite division of books, "some to be tasted, some to be chewed, and some few to be swallowed and digested," this present work is neither superficial enough to be accorded the first nor profound enough for the last. This is neither a reference book nor a textbook or manual but an honest plea for a better understanding of mental illness, coupled with suggestions for a community-minded drive toward better mental health. It suffers by comparison with Halliday's striking analyses but has perhaps the merit of "getting through" to a larger cross section of the interested reading public. Dr. Yost says nothing that the psychiatrist and psychologist does not already know and has not already said, but his approach is more in accord with the popularizing trend in scientific literature. The scientist may be mildly startled by such a folksy script writer technique as is seen at the beginning of chapter 1: "Our plane was speeding westward across the continent. Only a few short hours before, we had left the airport in New York, and we were already a short distance beyond Chicago, the Windy City. Glancing idly at a newspaper which a fellow passenger had offered to share with me, my eye fell on a headline that arrested my attention: 3,548,000 Babies Born Last Year. It was an official figure from the Bureau of the Census. I saw in my mind's eye those squirming, laughing, crying, cooing infants—a cross section of the America of tomorrow. Among them would be all sorts of babies—yellow, red, white, black; long babies, short babies, chubby and thin ones, all kinds and conditions of babies—our country's most precious resource."

Fortunately his mind's eye is rapidly cleared of the "chubby and thin ones" and he goes on to a clear and lucid exposition of mental function from topographical, dynamic, and economical viewpoints, with a wealth of apt illustration and example. He pays compliment to the roles of various important socioenvironmental factors—the mother, the teacher, and the community. This is followed by a necessarily concise but accurate discussion of psychopathology in terms that enlighten but do not in the process disturb. It is difficult to see why the more technical procedures of psychotherapy and psychosurgery are interpolated between psychopathology and discussion of such conditions as alcoholism, drug addiction, epilepsy, and suicide, but perhaps Dr. Yost was using the social impact of such phenomena to lead up to his final chapters on social responsibility and the interrelationship between psychiatry and religion. This book should do much to stimulate the desire to approach the literally awful problem of mental illness on the grand scale that is so urgently required. The suggestions that he makes are valuable and positive, and his book renders a definite service to community and national needs.

These book reviews have been prepared by competent authorities but do not represent the opinions of any official bodies unless specifically so stated.

Sexual Behavior in the Human Female. By Alfred C. Kinsey, Wardell B. Pomeroy, Clyde E. Martin, and Paul H. Gebhard, Research Associates, Institute for Sex Research, Indiana University, Bloomington. Cloth. $8. Pp. 842, with 155 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5; W. B. Saunders Company, Ltd., 7 Grape St., Shaftesbury Ave., London, W.C.2, 1953.

There are few works published in the last decade that have excited as much comment and argument as "Sexual Behavior in the Human Female." Because of this controversy THE JOURNAL *ignored its usual practice of submitting a book for review to one person; in this instance two persons, both widely known in their fields, have been asked for reviews.*—ED.

A great deal has been written and said about the Kinsey reports on the sexual behavior of the human male and female. For some reason the title of this book bears the earmark of a neologistic twist, for it reads: "Sexual Behavior *in* the Human Female." Attention is called to this not because a carping thrust at Kinsey and his collaborators is intended but rather to point up a certain aspect of Kinsey's thinking that interferes with the clarity of his scientific attitude. There is no doubt that Kinsey's general approach is scientific. There is no doubt that his approach suffers from a number of defects and that it also enjoys, in many respects, the credit of validity of any serious scientific undertaking. What seems lacking in Kinsey's approach, however, is a true awareness of the fact that we are dealing with a complex psychobiological phenomenon. Behavior in the human female is not a phenomenon but a semantic artifact. Behavior is a part of the human being, and it is the manifestation of a complexity called a personality. Neither biology nor medical psychology would place objective behavior within a person.

If Kinsey had deliberately and overtly left out of consideration the psychological aspects of the problem on which he worked, there would be reason for regretting such an omission, but one would not quarrel with the omission. It is the privilege of the research worker to limit his field of investigation in any manner he wishes. The trouble is that Kinsey does not leave out human psychology but deals with it almost in the manner of a nonscientific layman who tries to give authoritative voice to his prejudices. This is a defect of serious importance.

Kinsey thus states almost glibly: "Freudian hypotheses of psychosexual development proceeding, as a rule, from narcissistic (masturbatory) interests and activities to interests in other individuals whose bodies are similar (the homosexual interests), and finally to interests in individuals who are physically different (the heterosexual interests), are not substantiated by the preadolescent or adolescent histories of either the females or the males in the sample" (page 109). The above is not an accurate presentation of the Freudian hypotheses, nor does it include the central points of Freudian considerations—the evolution of the love affects that accompanies the evolution of psychosexual development, an evolution that culminates normally in object love and the striving toward parenthood. To omit love and the inner aspiration to become a parent is to omit that which makes the human (male or female) human. Leave love out of consideration and you have no sexual behavior, but only a manifestation of the physiological reaction of the sexual organs and their adnexa. Modern biology has no more right than modern medicine to exclude the human elements from the human being—for the totality of the psychobiological unit that is man is not overlooked today either by the practicing physician or by the medicopsychological research worker.

Then, too, one wonders how it happens that Kinsey feels justified in stating that the above-mentioned Freudian hypotheses "are not substantiated by the preadolescent or adolescent histories of either the females or the males in the sample." How did Kinsey go about verifying these hypotheses about the evolution of the human personality, which lies primarily in the sphere of the unconscious? It is as if one observed carefully a flight of birds and then stated that Newton's formulation about the laws of gravity is not substantiated in the sample of flying ducks.

There seems to be a lack of understanding of such psychosexual phenomena as "vaginal orgasm"—Kinsey shows the common tendency to look for specific innervations, which is not too hopeful an attitude from the standpoint of scientific understanding of the phenomenon. After all, in dealing with sexual issues we are considering a supercharged human instinct that is responsible for the greatest artistic inspirations and most heinous crimes in human history. And yet Kinsey looks upon it all with the simple eye of an old-fashioned behaviorist making rather trite and general references to learning and conditioning.

He seems to encourage masturbation (page 263) and overestimate the physiological, sensual orgasm without considering the psychological factors that might inhibit the healthiest neuromuscular apparatus and leave a sense of affective vacuum even after the most intense physiological orgasm. Clinically we may observe this in patients with neuroses and involution psychoses (particularly in women). The whole field of clinical considerations seems to escape Kinsey, and his apparent biases lead him to such striking statements as this, for instance: "It should, however, be emphasized again that most of the husbands who accepted or encouraged their wives' extra-marital activity had done so in an honest attempt to give them the opportunity for additional sexual satisfaction" (page 435). Of course the above statement is both sweeping and inconclusive, for it omits the consideration of human affects and overlooks the simple clinical fact that the alleged good will of the singularly permissive husbands in question is in itself a sign of a serious clinical condition with which the practicing psychiatrist is not unacquainted.

It is difficult to rid one's self of the impression that from the clinical, psychobiological point of view Kinsey's latest book is not very scientific and that he falls frequently into the same formalistic, pseudoscientific trap that characterized the classical Greeks and the Arabs like Avicenna, who considered love an illness because it produced a flushed face and an accelerated pulse. When Kinsey speaks of some "physiological effects," his approach is almost that of the ancients who disregarded love and overestimated the orgasm as an end in itself (page 171 or 166).

One cannot but admire the perseverance and tenacity with which Kinsey sticks to his work, but from the medicopsychological point of view he does dehumanize both the human male and the female. And he does this despite the best perspectives offered by modern medical psychology—all this even if we leave entirely out of consideration the question of traditional morality and sex.

(Second review)

The present volume constitutes the second progress report on the study of human sex behavior that has been under way for 15 years at the University of Indiana. Dr. Kinsey and his associates have had charge of this project, which has been supported by the Rockefeller Foundation through the Committee for Reşearch on Problems of Sex of the National Research Council. It has been a fact-finding survey in an attempt to discover what people do sexually, what factors account for their patterns of sexual behavior, how their sexual experiences have affected their lives, and what social implications there may be in each type of behavior.

The data in this volume are based on personal interviews with 5,940 white women from a total of almost 8,000 women who were interviewed. Much criticism has been directed against the selection of the persons who comprise this study, for they are not necessarily representative of our female population. The investigators acknowledge this defect in their material and note that a better sampling was not possible because they had to make use of those volunteers who presented themselves. They obtained detailed information about the lives of the women who were interviewed and their various sex practices. The questions concerned masturbation; nocturnal sex dreams; premarital petting; premarital, marital, and extramarital coitus; and homosexual responses. All of these forms of sexual gratification were brought together to provide a pattern of the total sexual outlet of the groups of women who were studied. The data are presented in a series of tables at the end of each chapter and illustrated by many simple graphs. The historical, social, moral, religious, and emotional implications are discussed fully. There are extensive references to the old and current literature, which are presented in footnotes throughout the text.

The last five chapters are devoted to the anatomy, physiology, and the neural mechanism of the sexual response and orgasm. The psychological and hormonal factors that may influence the sexual act are discussed. The male and the female are compared in these pages, and the authors suggest that there are no great differences between the sexes except that psychological factors probably play a more important role in men than in women. A comprehensive bibliography is included.

This report is a detailed, careful, scientific analysis of a fairly large sampling of our feminine population. Together with the report on the human male, many pertinent facets of human sexual behavior are presented. Although the groups investigated may not be representative of our population and the implications may not be valid for any one person or group of persons, the data are the best available at present. This book does not qualify as a sensational sex thriller but does represent a scientific report of a study still in progress, which is being diligently pursued by a group of experts well qualified to carry on this project. This book and its companion volume on the male should be read by physicians, educators, sociologists, and all others who are seriously interested in human sexual behavior.

Handbuch der inneren Medizin. Begründet von L. Mohr und R. Staehelin. Herausgegeben von G. v. Bergmann, W. Frey, und H. Schwiegk. Fünfter Band, Teile 1-3: Neurologie. Redigiert von R. Jung. Teil 1, bearbeitet von R. Brun, et al. Teil 2, bearbeitet von P. E. Becker, et al. Teil 3, bearbeitet von E. Bay, et al. Fourth edition. Cloth. 656 marks; subscription price, 524.80 marks. Pp. 1543, with 591 illustrations; 966, with 286 illustrations; 1531, with 388 illustrations. Springer-Verlag, Reichpietschufer 20, (1) Berlin W. 35 (West-Berlin); Neuenheimer Landstrasse 24, Heidelberg; Göttingen, 1953.

This section on neurology of the "Handbook of Internal Medicine" is within the great tradition of the German scientific handbooks. Although less extensive than Foerster's famous "Handbuch der Neurologie," it devotes about 3,000 pages to a discussion of clinical neurology and to its anatomic and physiological foundations. About 25 authors share in this work, which has been carefully edited by R. Jung. Each contribution is provided with an extensive bibliography listing monographs and special papers and with numerous figures. The quantity of these illustrations is indicated by the fact that the chapter on arteriography has 49 figures. In general, the whole field has been reviewed objectively, without eliminating the special flavor of individual articles that is based on the particular experience of the writer. The Anglo-American literature is extensively utilized. There is a detailed author and subject index, and the printing and binding are excellent. The work is highly recommended as an authoritative review of theoretical and clinical neurology.

Volume 1 contains essays on the general physiology of the nervous system; the general anatomy, physiology, and pathology of peripheral nerves and symptoms resulting from their disorders; the spinal cord; the autonomic nervous system; and the brain. The second volume describes neurological methods (clinical and laboratory); medical and surgical therapy; and the diseases of peripheral nerves, spinal cord, autonomic nervous system, and muscles. This volume also includes a special article on multiple sclerosis. The third volume deals with circulatory disturbances and inflammatory diseases of the brain; syphilis, trauma, and tumors; diseases of the brain stem, extrapyramidal system, and cerebellum; epilepsy and related conditions; and symptomatic psychoses and neuroses.

The Symptoms and Treatment of Acute Poisoning. By G. H. W. Lucas, Professor of Pharmacology, University of Toronto, Toronto, Ontario. Cloth. $4. Pp. 308. The Macmillan Company, 60 Fifth Ave., New York 11, 1953.

This book was written to provide practicing physicians with a ready reference for treating patients who have been poisoned. The subjects considered include supplies and apparatus, emetics and gastric lavage, poisonings in children, collections and preservation of samples for analysis, useful drugs and preparations, and cases of poisoning. A most noteworthy addition is the chapter on poisonings in children, since this always presents a special problem to the physician. In addition a table of approximate equivalents, domestic measures, etc., and a table of fatal doses are included. The book is complete as to the number of toxicologic agents discussed. The index is easy to consult. Trade names are used along with the generic names, and in the index the trade names have been placed in italics together with an abbreviation for the name of the manufacturer. The book can also be used as a practical handbook on the symptoms and treatment of acute poisoning by dentists, nurses, and pharmacists. This is a useful addition to the literature of toxicology.

Side Effects of Drugs. By L. Meyler. Translated by Ph. Vuijsje and W. Mulhall Corbet. Cloth. $5.50. Pp. 268. Elsevier Press, Inc., 402 Lovett Blvd., Houston 6; 155 E. 82nd St., New York 28; 118 Spuistraat, Amsterdam C, Netherlands, 1952.

This reference book lists the side-effects that may occur after the therapeutic administration of drugs. It provides the practicing physician with a handy and somewhat extensive guide to determine whether unexpected symptoms are the result of the disease or are actually the side-effects of the drug. The book includes most compounds in common use. The drugs are classified according to their therapeutic properties. Two special chapters are devoted to metals and metalloids and one to serums and vaccines. The book is well documented and contains an extensive bibliography at the end of each chapter. The index is well arranged and complete. The author has included very little critical evaluation of the side-effects discussed. That the incidence of occurrence of the side-effects is not indicated is another drawback. In this age of antibiotics and chemotherapy, however, this handy reference should prove of unestimable value.

Influenza and Other Virus Infections of the Respiratory Tract. By C. H. Stuart-Harris, M.D., F.R.C.P., Professor of Medicine, University of Sheffield, Sheffield, England. With foreword by C. H. Andrewes, M.D., F.R.C.P., F.R.S., Deputy Director, National Institute for Medical Research. Cloth. $6. Pp. 235, with 98 illustrations. [Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2]; Edward Arnold & Co., 41-43 Maddox St., London, W.1, 1953.

Acute viral infections of the respiratory tract are a greater problem than ever in clinical practice because of the success of antibiotics and sulfonamides in overcoming bacterial infections. In this book a distinguished English authority successfully differentiates what is known about influenza, virus pneumonia, and other common respiratory diseases from what is still nebulous and vague. Influenza receives the most attention, both because of its importance as a potential killer and because of knowledge already gained by concerted clinical, epidemiological, and laboratory investigations. In addition, there are several informative and instructive chapters on the common cold, febrile catarrh, atypical pneumonia, psittacosis and ornithosis, Q fever, and laboratory diagnosis. This book contains much valuable information and is highly recommended to specialists and general practitioners alike.

Advances in Virus Research. Volume I. Edited by Kenneth M. Smith and Max A. Lauffer. Cloth. $8. Pp. 362, with illustrations. Academic Press, Inc., 125 E. 23rd St., New York 10, 1953.

The effective dissemination of the results of virus research has suffered greatly through the lack of journals primarily devoted to this field. This is particularly true with fundamental investigations of the nature and immediate characters of the viruses themselves, the reports of which are scattered widely in publications not often consulted except by specialists in the basic sciences. In this volume an attempt is made to present each year timely reviews of progress in the study of those properties of viruses that establish their place in the biological, physical, and chemical worlds. The first volume contains an absorbing account of the insect viruses effectively illustrated by electron micrography by G. H. Bergold. A fertile field of contemporary research is that of virus multiplication. The work of this character on bacteriophages is reviewed by Epstein, and that with the influenza virus is considered exhaustively by Henle. The general problems and results of virus purification are discussed by Sharp. The properties and functions of nucleic acids in viruses are described by Markam. The phenomenon of interference between viruses resulting in protection against or suppression of infection of one by the other is reviewed by Bennett, and the transmission of plant viruses by insect vectors, the leaf hoppers, is recounted by Black. A comprehensive account of the properties of the poliomyelitis virus relative to types, culture, growth, immunologic properties, and gamma globulin prophylaxis is given by Melnick. The reviews are excellent, and the series of publications within a few years should provide a highly concentrated source of the principal findings in the nature and properties of viruses.

QUERIES AND MINOR NOTES

DISCOID LUPUS ERYTHEMATOSUS

To the Editor:—*A married woman, aged 37, has had discoid lupus erythematosus for 15 years. She received treatment at several clinics, all to no avail. Quinacrine (Atabrine) therapy was begun on April 10, 1953, and the dose was increased from 50 to 150 mg. daily. All lesions blanched and the subjective symptoms, such as stinging and burning, disappeared. There have been occasional short attacks of nausea, vomiting, and diarrhea. She is now taking a maintenance dose of 75 mg. of quinacrine daily, and, while there has been a moderate return of redness to the lesions, there has been neither burning nor induration. How long may this maintenance dose be given?*

George M. Kesl, M.D., Port Huron, Mich.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—In general, in an attempt to avoid toxic reactions in quinacrine (Atabrine) therapy of discoid lupus erythematosus, a rest period is given sometime during the course of treatment. The weight loss, diarrhea, and nausea in this patient are reactions to quinacrine, although not serious ones. It is recommended that the maintenance therapy be discontinued for three to four weeks if the skin lesions are doing well. The blood cell counts and urinalyses should be done even during this rest period, for sometimes the toxic reactions may be delayed. Chloroquine diphosphate is less toxic than quinacrine and may be substituted for quinacrine at the end of this rest period in a dosage of 250 mg. twice a day or even only once a day for a period of several months. If the skin lesion is under good control, then all therapy may be stopped unless a flare-up occurs. If the patient's lesions usually flare-up in spring, then a preventive routine of 250 mg. of chloroquine diphosphate may be started early in March. If an active flare-up occurs, 250 mg. four times a day may be given. Routine blood cell counts and urinalyses should be done also in chloroquine diphosphate therapy.

Answer.—The drug may be given as long as necessary if the patient is observed at frequent intervals for signs of intolerance. Perhaps it would be well to change to chloroquine which is less toxic than quinacrine, beginning with 0.25 gm. three times daily for a while then reducing to an effective maintenance dose.

HIRSUTISM AT THE CLIMACTERIC

To the Editor:—*I have seen two cases of lanugo-type hair on the faces of two women, aged 40. One apparently had a short period of functional bleeding and the other was approaching the climacteric. Could you suggest a differential diagnosis?*

M.D., Maine.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—It has long been thought, and with good evidence, that the pilary system is controlled by the endocrine glands such as the gonads, thyroid, anterior pituitary, and adrenal cortex. It is not surprising therefore that some hirsutism should appear at the time of the climacteric. Idiopathic hirsutism of a permanent character has also been observed after puberty and pregnancy as a genetic characteristic and after diseases such as mumps, encephalitis, neuritis, and multiple sclerosis. Certain drugs such as diphenylhydantoin (Dilantin) and testosterone may produce transient hirsutism. Since there was no evidence of any of these factors nor of endocrine gland tumors in either of these two cases, it may be assumed that the hairs are associated with the menopause.

Answer.—It is not uncommon for facial hirsutism to develop in women approaching the climacteric. The mechanism of this excessive hair growth is not very well understood. Diminution of estrogen secretion by the ovaries may be responsible for it; however, the administration of estrogen does not influence the hair growth to any marked degree. Estrogen-containing facial creams have been used with some slight success. Apparently, the androgens secreted by the adrenal cortex control hair growth, and it is difficult to influence the secretion of these androgens. In a differential diagnosis of the condition, the investigation should center around the function of the adrenal cortex, and a 17-ketosteroid determination would reveal whether there is excessive androgen secretion. Not uncommonly a slight or even moderate increase in excretion of 17-ketosteroids is found in this condition. Only when a great excess is found should procedures aimed at removing portions of the adrenal cortex be considered. Another theory regarding the development of hair growth in women implicates the theca cells of the ovary, which elaborate an androgen. This, however, is speculative at the present time, and it is only in cases of arrhenoblastoma that ovarian tissue is known to elaborate androgens.

NUMBNESS OF HANDS IN PREGNANCY

To the Editor:—*I frequently encounter pregnant women who complain of numbness and tingling of the hands. Others complain of a painful stiffness of the fingers, especially in the morning, that improves with use during the day. At times there is a slight swelling of the hands and temperature changes are noted. These patients all have had adequate calcium and vitamin intake, and the sodium and water balance has not been deemed a factor. All symptoms have spontaneously disappeared during the puerperium. What is the cause and treatment of this condition?*

Fred S. Taber, M.D., New Brunswick, N. J.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—Stiffness of the joints and slight swelling of the hands are not unusual in pregnancy and during the premenstrual period. They are due to the sodium retention that attends these states, and accordingly they ordinarily respond to sodium restriction or ammonium chloride. There is no good explanation for the more troublesome paresthesias of the hands that are occasionally seen during pregnancy, and these usually follow the distribution of the ulnar nerve. Large doses of vitamins, especially the vitamin B complex, have been given without notable effect. Salt is restricted and ammonium chloride given (often with indifferent effect) on the possibility that edema at the nerve roots may be the cause. The patient who complains of continuous burning, aching pain that precludes the use of the hands should be suspected of having a serious underlying vascular or neurological disturbance and should be investigated with this in mind. Such symptoms are not among the changes of normal pregnancy.

Answer.—Numbness and tingling of the hands during pregnancy are forms of neuritis, the exact cause of which is not known. The condition disappears shortly after delivery. In many cases no treatment is necessary, but moist or dry heat, salicylates, codeine, and niacin may be helpful. The treatment of this form of neuritis is identical with that in nonpregnant patients.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically so stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

POLIOMYELITIS AT A SUMMER CAMP

To the Editor:—*I am chairman of the medical committee of a summer camp for children. The camp consists of a common dining room and a series of bunks. In the event one of the campers contracts poliomyelitis, should all the children be quarantined or only those who resided in the same bunk as the ill camper? Should any campers be allowed to return home? When should other children be allowed to come in as new campers? What period of time should be allowed later for adults to come to camp as campers?*

M.D., Ohio.

Answer.—The child stricken with poliomyelitis may be removed to a hospital with the consent of the proper health authorities. All other children should be confined to the camp for two weeks. There should be daily medical inspection, and, if any evidence of illness develops, the patient should be isolated. New children should not be accepted at the camp. All those at the camp should be advised to remain. It is generally believed the danger of poliomyelitis is not diminished by leaving a camp where the disease has occurred. Local health regulations may interfere with children returning to their homes. No new campers should be admitted until at least two weeks have elapsed since the last case of poliomyelitis developed. It would be advisable to allow a period of one week to elapse after all children had left the camp before admitting adults. During that time a thorough sanitary inspection should be made of the premises. Food supplies, water, milk, and insects should receive consideration. The foregoing advice is based largely on Recommendations of the Illinois Department of Public Health issued in June, 1953.

CHRONIC ULCERATIVE COLITIS

To the Editor:—*A 19-year-old girl has had chronic ulcerative colitis for several years. Eight months ago she was operated on for an anal fistula. Since the operation, there has been practically no healing in the perineal wound despite adequate drainage and no connection with the anal canal or rectum. Suggestions of means to promote wound healing in this case will be appreciated.*

M.D., New York.

Answer.—The fistulous tract originally must have been connected with the lower bowel that was the site of chronic ulcerative colitis. Operative repair often involves the failure of the wound to heal because of infected material constantly laving the anal area and because of impaired vitality of the tissue. Clinical remission of chronic ulcerative colitis does not necessarily imply subsidence of the pathological process in the colon. This can be readily determined by mucosal aspiration wet smear, using a heavy bore capillary tube inserted through the sigmoidoscope. A purulent cytological material in the mucosal exudate is indicative of active intramural infection. Cutting into or through such an infected area involves the hazard of poor healing, since drainage is not merely of the former fistulous tract but of the colon itself. In some instances, fecal material may be seen in the wound because of a persistent opening in the rectum or anal canal. Operative interference should be avoided, if at all possible, for perianal conditions complicating chronic ulcerative colitis. Ischiorectal abscess is infrequent but usually must be treated surgically.

The following treatment is suggested for this patient: (1) good supportive therapy, including a high vitamin, high protein diet; (2) administration of 250 to 500 mg. of ascorbic acid three times a day; (3) microcrystalline sulfathiazole powder sprinkled into the wound once or twice daily after it has been cleaned with 50% hydrogen peroxide; and (4) active immunization against the flora recovered from the wound, usually coli and enterococcus organisms. If these measures fail after 10 weeks, a short circuiting of the fecal stream (ileostomy) usually has to be done. After a second 10 week period, failure to heal may necessitate a total colectomy. Ileostomy or subtotal or total colectomy should not be undertaken lightly; they are by no means absolute solutions in chronic ulcerative colitis. On the other hand, a number of patients, such as the girl described in the query, have improved after local administration of microcrystalline sulfathiazole and conservative supportive therapy.

SMALL TUMOR NEAR HYOID BONE

To the Editor:—*A 50-year-old man suddenly discovered a small tumor in the region of the hyoid bone a bit to the right. It is about 1 in. in diameter and hard, but is asymptomatic. The diagnosis is probably a thyroglossal cyst. Do you advise removal of the cyst? What symptoms and signs could develop?*

M.D., Rhode Island.

Answer.—The most likely diagnosis of a round midline or near midline lesion at the level of the hyoid bone is thyroglossal cyst. It may appear at any age but usually appears in patients younger than 50. The commonest complication of thyroglossal cyst is obstruction. The mass could be an adenoma of the thyroglossal tract or even a carcinoma. There are lymph nodes in this vicinity, and it could be metastatic from a primary tumor in the thyroid below. It is quite unlikely that the roentgenogram will reveal anything significant. Since its diagnosis is uncertain, the removal of the tumor should be advised.

HAND-SCHÜLLER-CHRISTIAN DISEASE

To the Editor:—*A girl with Hand-Schüller-Christian disease died at age 4 after about three years of illness. There are two healthy siblings, aged 8 and 10. The parents want another child but are afraid it might be affected. Is Hand-Schüller-Christian disease hereditary?*

Hubert W. Coleman, M.D., Avon Park, Fla.

Answer.—There is no evidence that lipid histiocytosis of cholesterol type (Hand-Schüller-Christian disease) is hereditary. The odds of another child being affected are so small that they do not represent an appreciable risk.

GLUCOSE SOLUTION INTRAMUSCULARLY

To the Editor:—*I would like to comment on the answer to the query on the administration of glucose solutions intramuscularly on page 100 of* The Journal, *Jan. 2, 1954. While sloughing will not occur if 5 or 10% glucose solutions are given intramuscularly, a temporary shift of electrolytes and some fluid into the area of injection will occur. Because of the resulting lowered electrolyte concentration in the extracellular compartment, a further transfer of water to the intracellular phase will occur. These shifts result in a depletion of the plasma and interstitial fluid that may lead to serious consequences.*

It has been shown that hypotension and oliguria may occur as a result of the decrease in the plasma volume (10 to 30%) that follows hypodermoclysis of 2,000 ml. of a 5% glucose solution in distilled water. Therefore, I feel it especially undesirable and dangerous to give glucose in water in amounts up to 80 ml. per kilogram of body weight, particularly when the patient is dehydrated or if hyaluronidase is employed. Since a considerable decrease in the plasma volume has been noted after 35 to 40 ml. of glucose in water has been given per kilogram of body weight by hypodermoclysis to normal patients, it would seem unwise to administer electrolyte-free fluids to dehydrated patients by this method. While hyaluronidase causes the fluid to spread, or diffuse, throughout a larger area, relieving pain and causing a more rapid absorption, it also accelerates the shift of electrolytes. Thus, although the undesirable shift of fluid that leads to a diminution in the plasma volume is of shorter duration, its effect is more profound when hyaluronidase is employed. For a better understanding of the dangers of administering electrolyte-poor fluids by hypodermoclysis, the following references should be consulted (Surgery **52**:*305, 1952; Surgical Forum: Proceedings of the Forum Sessions of the 37th Clinical Congress of the American College of Surgeons, 1952, Philadelphia, W. B. Saunders Company, 1952, pp. 559-564;* J. A. M. A. **150**:*942 [Nov. 1] 1952 and* J. A. M. A. **151**:*644 [Feb. 21] 1953).*

William E. Abbott, M.D.
University Hospitals
2065 Adelbert Rd.
Cleveland 6.